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The Autonomic Nervous System in Relation to the Thoracic Viscera*

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ANATOMICAL RELATIONSHIPS

The thoracic viscera are innervated through both the sympathetic and the parasympathetic divisions of the autonomic nervous system. The sympathetic nerves in question arise from the cervical and thoracic sympathetic trunk ganglia. The preganglionic components of these nerves emerge from the spinal cord via the thoracic spinal nerves, traverse the white communicating rami of these nerves and effect synaptic connections in the sympathetic trunk ganglia (Figs. 1 and 2). The ganglion cells involved in the parasympathetic innervation of the thoracic organs are located in the ganglia of the cardiac and pulmonary plexuses and the intramural ganglia of the respiratory tract and the esophagus. The preganglionic components of the parasympathetic nerves emerge from the brain stem via the vagus nerves. Visceral afferent spinal nerve fibers reach the thoracic viscera via the sympathetic nerves. Visceral afferent components of the vagi which supply the thoracic viscera likewise are associated with the parasympathetic nerves (Figs. 1 and 2).

The cardiac plexus consists of a superficial and a deep portion. The superficial cardiac plexus lies mainly superficial to the pericardium in the concavity of the arch of the aorta. It receives fibers principally via the left superior cervical sympathetic cardiac nerve and the inferior cervical cardiac branch of the left vagus. It includes a small ganglion, the cardiac ganglion of Wrisberg. The deep cardiac plexus consists of two lateral portions joined together by numerous bundles of nerve fibers. The right portion receives fibers via the right superior, middle and inferior cervical and the thoracic

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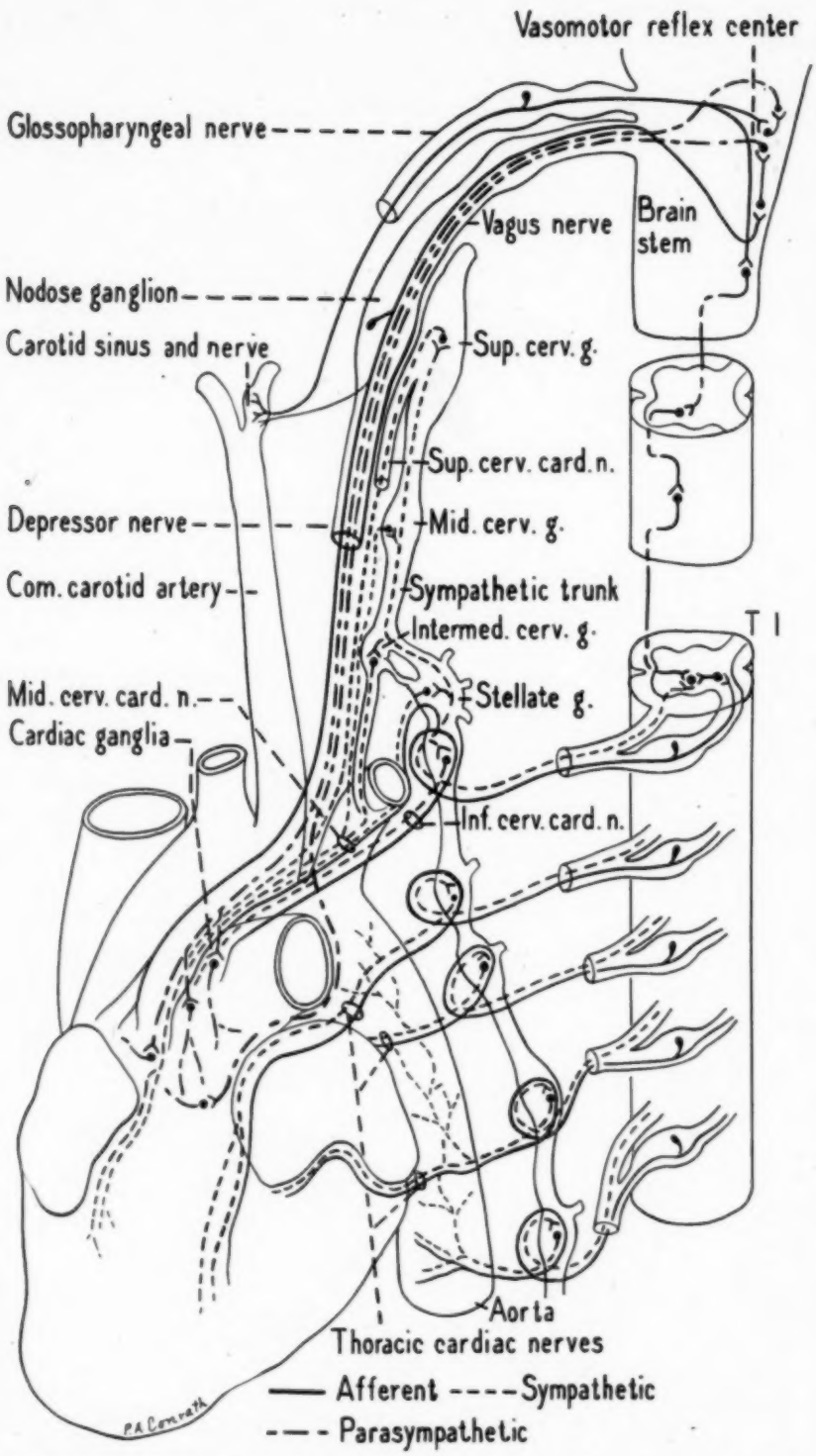

Fig. 1—Diagrammatic illustration of the sympathetic, parasympathetic and afferent innervation of the heart.

sympathetic cardiac nerves and all the cardiac branches of the right vagus. The left portion receives fibers via the left middle and inferior cervical and the left thoracic cardiac nerves and the superior cervical and thoracic branches of the left vagus. It also receives communications from the superficial cardiac plexus.

The anterior (right) coronary plexus is made up mainly of fibers derived from the superficial cardiac plexus and the right portion of the deep cardiac plexus. It supplies the right coronary artery and the adjacent cardiac tissues. The posterior (left) coronary plexus is derived mainly from the left portion of the deep cardiac plexus and receives some fibers from the right portion of the deep plexus. It supplies the left coronary artery and adjacent portions of the heart. The efferent innervation of the coronary vessels includes both sympathetic and parasympathetic fibers, but the former are much more abundant than the latter (Nettleship[1]).

The deep cardiac plexus includes numerous small ganglia most of which are located in the subepicardial connective tissue. Although some lie deeper than others, various investigators including Perman,[2] Woollard,[3] and Francillon[4] observed none which could be regarded as intramuscular. They are distributed mainly on the posterior surfaces of the atria in relation to the great vessels (Perman[2]). Few or none are located on the ventricular side of the atrioventricular groove. The axons of the cardiac ganglion cells terminate in relation to the musculature of the atria and the coronary vessels and the atrioventricular conducting system. Sympathetic fibers innervate the entire myocardium, the atrioventricular conducting system and the coronary vessels.

The heart is abundantly supplied with afferent nerve fibers. Receptors, particularly in the subendocardial tissue, have been described by various investigators including Smirnow,[5] Dogiel,[6] Michailow,[7] and Woollard.[3] In methylene blue preparations of the cat's heart, Nettleship[1] described an endocardial plexus the afferent nature of which is demonstrated by the observation that it undergoes extensive degeneration following section of the vagus nerves distal to the nodose ganglia, but not following section of these nerves proximal to the nodose ganglia. Ablation of the spinal ganglia in the upper thoracic segments resulted in no extensive degeneration of the endocardial plexus except near the apices of the ventricles. Nettleship also described a plexiform structure surrounding the basal portions of the aorta and the pulmonary artery which lies in part adjacent to the adventitia of these vessels and is continuous with the subepicardial network at the base of the heart but quite distinct from the endocardial plexus. It is made up mainly of vagus afferent fibers. In methylene blue preparations of the rat's heart, King[8] described receptive end organs in the myocardium.

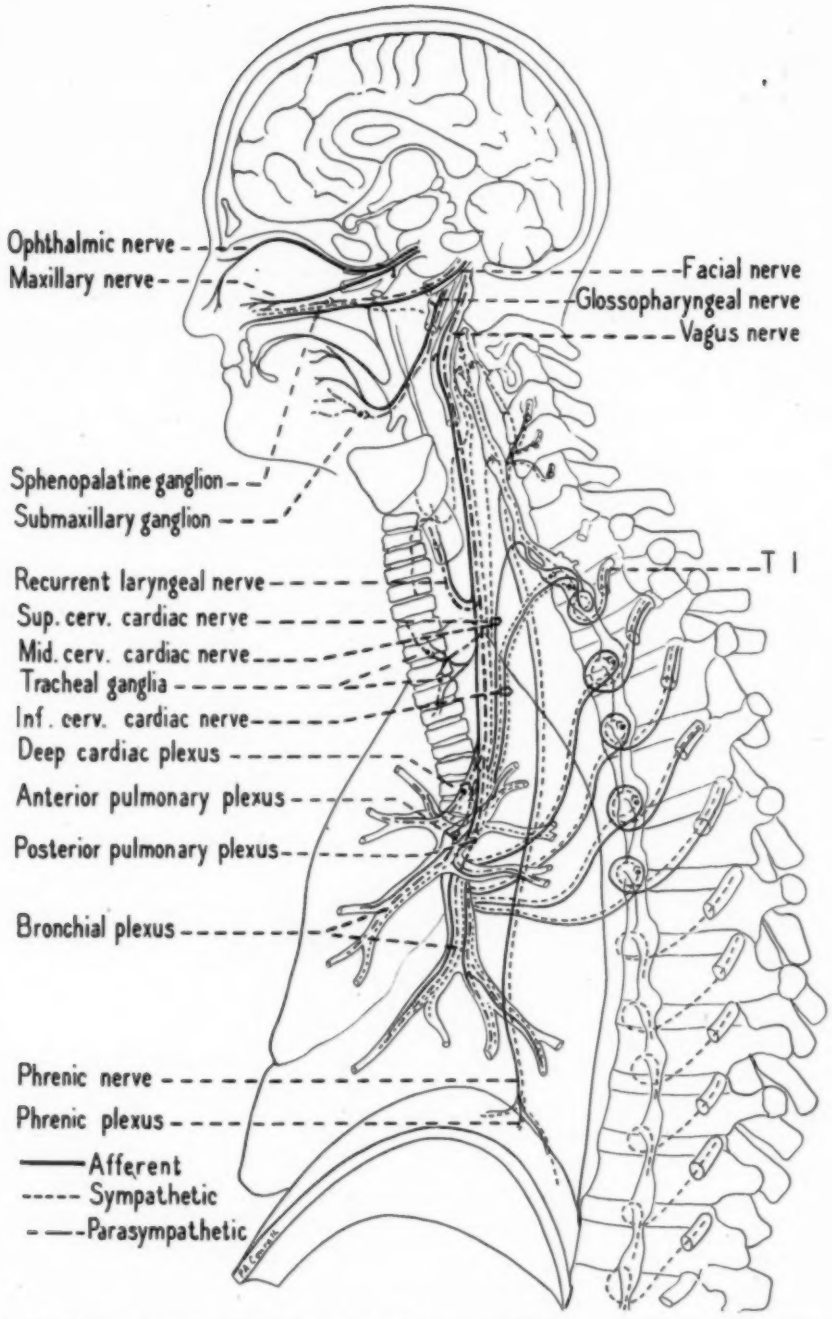

Fig. 2—Diagrammatic illustration of the sympathetic, parasympathetic and afferent innervation of the respiratory tract.

The coronary vessels also are abundantly supplied with afferent nerve fibers. Unlike most of the afferent nerve fibers which terminate within the heart, these are not appreciably affected by section of the vagi distal to the nodose ganglia, but undergo extensive degeneration following ablation of the spinal ganglia in the upper thoracic segments (Nettleship[1]). The major portion of the afferent innervation of the coronary vessels obviously is effected through spinal nerve components.

The trachea and the esophagus are innervated through both sympathetic and parasympathetic nerves. The former are derived directly from the sympathetic trunks; the latter comprise preganglionic components of the vagus nerves and intramural ganglion cells. These organs also are supplied with afferent fibers of spinal ganglion origin via the sympathetic nerves and with afferent cranial nerve components mainly through the vagi.

The Pulmonary Plexuses—The innervation of the lungs involves the pulmonary plexuses, which comprise an anterior and a posterior plexus on either side (Fig. 2). The anterior pulmonary plexus lies in relation to the anterior aspect of the root of the lung. It is joined on both sides by fibers derived from the corresponding part of the deep cardiac plexus and on the left side also by fibers from the superficial cardiac plexus. The posterior pulmonary plexus is located in relation to the posterior aspect of the root of the lung. It is larger than the anterior plexus and is made up mainly of branches of the vagus nerve and rami arising from the second, third and fourth thoracic sympathetic trunk ganglia. Nerves extending distalward from the pulmonary plexuses are distributed to all parts of the lungs (Fig. 2). These nerves include, in addition to the efferent autonomic fibers, afferent components both of spinal and of vagus nerves; but the afferent innervation of the bronchial system is predominantly vagus.

Receptive end organs in the trachea and bronchi have been described in detail, particularly by Larsell[9] and Larsell and Dow.[10] According to their accounts, the tracheal and bronchial mucous membranes are abundantly supplied with receptors, many of which lie in intimate relation to the epithelium. They are present throughout the respiratory tract including the atria. Elftman[11] also described receptive end organs in relation to the alveolar ducts and alveoli. Larsell and Dow also described muscle spindles in the bronchial musculature.

PHYSIOLOGICAL RELATIONSHIPS

The Cardiac Nerves.—Although cardiac muscle has the inherent capacity for rhythmic contractions, cardiac activity is regulated through neural and hormonal mechanisms. In general, sympathetic

stimulation elicits cardiac acceleration, whereas parasympathetic stimulation elicits cardiac inhibition. In a limited measure cardiac acceleration may be brought about by a reduction in parasympathetic tonus, and retardation of the cardiac rhythm by a reduction in sympathetic tonus, but the basal heart rate is not markedly modified by section of the sympathetic or the parasympathetic cardiac nerves. In experiments reported by Murphy,[12] the near basal heart rate of normal resting unapprehensive dogs which had been without food for twelve hours was not appreciably changed following bilateral extirpation of the upper five thoracic segments of the sympathetic trunk, including the stellate ganglion.

The existence of cardiac accelerator fibers in the vagus nerves of the dog has been demonstrated experimentally.[13] These fibers cannot be excited reflexly by stimulation of the carotid sinus or afferent vagus fibers, but respond promptly to acute cerebral anemia. Their threshold of stimulation is somewhat higher than that of the cardioinhibitors. They probably play no significant role in cardiac regulation under normal physiological conditions.

The latent period of the sympathetic cardiac nerves is longer than that of the vagi, but the effect of sympathetic stimulation continues longer than that of parasympathetic stimulation. Simultaneous stimulation of both the sympathetic and parasympathetic cardiac nerves elicits first cardiac inhibition and later cardiac acceleration.

Stimulation of the sympathetic cardiac nerves on the right side elicits greater acceleration of the cardiac rhythm than equal stimulation of the sympathetic cardiac nerves on the left side. Sympathetic stimulation on the right side also augments the force of the atrial contraction, but has no marked effect on the force of the ventricular contraction. Stimulation of the left sympathetic cardiac nerves, on the contrary, exerts no marked influence on atrial contraction but augments the force of ventricular contraction and tends to shorten the interval between atrial and ventricular contractions.

Afferent impulses arising in any part of the body may influence cardiac activity reflexly, but those arising in the carotid sinus and the cardio-aortic pressoreceptive and chemoreceptive zones are of major importance. The heart is protected against variations in arterial pressure and blood supply not only by depressor reflexes initiated in the cardio-aortic pressoreceptive area but also through other pressoreceptive mechanisms. The coronary blood flow depends in a large measure on the pressure in the aorta. The pressures in the left atrium, the left ventricle and the aorta depend mainly on the pressoreceptive sensitivity of the left ventricle, the aortic arch and the pulmonary artery. The venous pressure and the pressure in

the pulmonary artery and, consequently, the pressure in the right atrium and the right ventricle are regulated at least in part by the pressoreceptive sensitivity of the pulmonary arteries and veins, the venae cavae and the right atrium (Heymans[14]).

In a comprehensive review of the literature bearing on the regulation of the coronary circulation, Anrep[15] pointed out that the weight of evidence favors the hypothesis that constriction of these vessels is mediated through the parasympathetic nerves; their dilatation through the sympathetic nerves. This hypothesis has been supported by the results of studies reported by certain later investigators including Green,[16] Danielopolu and Margou,[17] Gollwitzer-Meier and Kruger,[18] and Bartschi.[19] In experiments reported by Kountz, Pearson and Koenig,[20] vagus stimulation resulted in retarding the rate of contraction of the normal human heart and increasing the flow of blood through the coronary vessels; sympathetic stimulation resulted in accelerating the heart rate and reducing the coronary flow. In perfusion experiments on the revived human heart, vagus stimulation resulted in reducing the coronary flow and sympathetic stimulation in increasing it, while there was dissociation of atrial and ventricular contractions and the rate of contraction was not influenced by nerve impulses. The action of drugs which, in the beating heart, increase muscular activity and decrease coronary flow, simulated the effects of sympathetic stimulation. The action of drugs which, in the beating heart, decrease cardiac muscle tonus and cause an increased coronary flow, simulated the effects of vagus stimulation. These results seem to support the conclusion that in man the cardiac nerves exert their influence on the flow of blood through the coronary vessels mainly through changes in the tonic state of the cardiac muscle. The results of experiments carried out on the dog's heart in a state of ventricular fibrillation, reported by Katz and Jochim,[21] support the assumption that the parasympathetic innervation of the coronary vessels includes only cholinergic vasodilator fibers which are tonically active, while the sympathetic innervation of these vessels includes both cholinergic vasodilator and adrenergic vasoconstrictor fibers. The action of the sympathetic nerves is predominantly vasoconstriction. According to this point of view the innervation of the coronary vessels is functionally comparable to that of other vessels which have both a sympathetic and a parasympathetic nerve supply.

At the beginning of muscular exercise, as observed by Green,[22] the flow of blood through the coronary vessels is sharply augmented and the coronary dilatation persists into the after period. This reflex coronary dilatation associated with muscular activity probably is a major factor in the nutrition of the heart during the added strain incident to the activity of skeletal muscles.

In experiments reported by Essex, Herrick, Baldes and Mann,[23] the effects of exercise were essentially similar on sympathectomized and normally innervated hearts. In both series of animals muscular exercise elicited increases in pulse rate, coronary flow and blood pressure. Section of the vagus nerves affected the responses of the heart to exercise more profoundly. Following bilateral vagus section, the heart rate increased less markedly due to muscular exercise than in animals with intact cardiac innervation regardless of whether the sympathetic cardiac nerves had been severed or remained intact. In animals with vagotomized or totally denervated hearts the coronary blood flow seemed to be influenced chiefly by the blood pressure.

The Pulmonary Nerves.—In general, stimulation of the parasympathetic nerves to the bronchi elicits contraction of the bronchial musculature, and stimulation of the sympathetic nerves elicits inhibition. Under certain conditions powerful stimulation of the sympathetic nerves may result in bronchoconstriction. Conversely, powerful parasympathetic stimulation sometimes results in bronchodilatation. On the basis of results obtained in experiments carried out on isolated perfused lungs of the guinea pig, Hebb[24] advanced the opinion that the sympathetic innervation of the bronchial musculature includes some cholinergic bronchoconstrictor fibers, at least in some animals. In Hebb's experiments bronchoconstriction elicited either by nerve stimulation or by the administration of acetylcholine was suppressed by adrenin, ergotoxine or atropine in appropriate doses.

Knowledge of the neural regulation of the pulmonary blood vessels is derived mainly from experimental investigations. Data reported particularly by LeBlanc and van Wijngaarden,[25] Berry and Daly,[26] Daly and von Euler,[27] Franklin,[28] and Hebb[24] support the conclusions that the sympathetic innervation of the pulmonary vessels includes the vasoconstrictor fibers and some vasodilator fibers, but the parasympathetic innervation also includes vasodilator fibers. In general the effect of the vasoconstrictors is more marked than that of the vasodilators. Pulmonary vasoconstriction or vasodilatation may take place independently of changes in the caliber of the bronchi; consequently, it may be assumed that, under physiological conditions, the pulmonary circulation is subject to regulation through the vasomotor nerves. In view of the mechanical conditions which obtain, it must be apparent that the pulmonary circulation is markedly influenced by the systemic circulation.

Regulation of Respiration.—Respiration is a complex visceral function the neural regulation of which involves not only autonomic nerves but also extensive automatic somatic neural mechanisms. Extensive consideration of the regulation of respiration is not

within the scope of this paper, but the trend of current teaching may be briefly indicated. The theory of the chemical control of respiration by the respiratory center in the brain stem has long been dominant, but during the past decade reflex factors have claimed increasing attention. The weight of experimental evidence now favors the assumption that chemical stimulation of the respiratory center is effected mainly through chemoreceptive mechanisms, particularly those associated with the carotid and aortic bodies. Direct chemical stimulation of the respiratory center is not precluded, but this center probably is less sensitive to some of the normal chemical stimulants than the peripheral chemoreceptors. The effectiveness of the latter has been amply demonstrated experimentally.

CLINICAL CONSIDERATIONS

Cardiac Disease—Pain is a common symptom of cardiac disease. The genesis of cardiac pain is not within the scope of the present discussion. Its vascular origin is indicated by its frequent occurrence following exertion and in association with a fall in blood pressure and the facts that it may be precipitated in many cases by injection of adrenin and may be controlled with nitrites (Jessen[29]). A causative relationship to cardiac pain of local anoxemia due to inadequate coronary circulation is strongly suggested by the observation of Boland[30] that such pain may be relieved in many cases by inhalation of pure oxygen. The prevalence of cardiac pain on the left side undoubtedly is related to the fact that the required volume flow of blood in the left coronary vessels is greater than that in the right, due to the greater volume of muscle to be supplied by the left coronary artery and the greater amount of work to be accomplished by the left ventricle than by the right. Coronary thrombosis, furthermore, occurs more frequently on the left side than on the right.

The nerve fibers which conduct impulses of pain from the heart are mainly visceral afferent components of thoracic spinal nerves, which reach the heart via the sympathetic cardiac nerves. Afferent vagus nerve components probably do not conduct impulses of pain from the heart. Relief of cardiac pain by block or section of the sympathetic cardiac nerves may be explained most satisfactorily on the basis of interruption of the pain conducting pathways. Interruption of the efferent pathways involved in coronary vasoconstriction may be a significant factor in improving the coronary circulation in the absence of significant pathology of the coronary vessels.

Pulmonary disease not uncommonly is accompanied by changes in the functional balance between the sympathetic and the parasympathetic nerves. In an extensive study of tuberculous patients,

Deutsch and Hoffmann[31] found parasympathetic tonus heightened during the second and third stages of the disease. The hectic flush, common in the later stages of tuberculosis, but which usually does not appear early in the disease, may be regarded as an expression of parasympathetic hypertonus. The relatively slow heart rate frequently observed during periods of fever, as compared with the heart rate during periods of the same degree of fever in other diseases, also indicates exaggerated parasympathetic tonus. A shift in the autonomic balance in the same direction also is indicated by the gastric hyperacidity which commonly occurs early in the course of tuberculous disease. Not infrequently the hyperacidity is associated with gastrointestinal hypermotility which results in nausea and a tendency to vomit. In some cases exaggerated parasympathetic tonus also results in spastic constipation.

In pulmonary tuberculosis, according to Pottenger,[32] sympathetic tonus may be increased due to central stimulation and the reflex effects of the inflammation in the lungs; consequently, sympathetic tonus may predominate. As the acute toxemia subsides the central sympathetic stimulation also subsides and in most cases exaggerated parasympathetic tonus again becomes evident. The patient's appetite is improved and his digestive capacity is increased. The associated hyperacidity usually is not sufficient to cause discomfort, but it may actually cause gastric distress. As the disease advances and toxemia and depressive emotional states become more marked, stasis and constipation and impairment of digestion also become more pronounced. The gastrointestinal symptoms usually observed during the later stages of tuberculous disease are less suggestive of parasympathetic tonus than those commonly observed earlier in the course of the disease. In those cases in which parasympathetic tonus clearly predominates during the later stages of the disease, it may be due, as suggested by Stammler,[33] to depression of the sympathetic tonus by the effects on the sympathetic ganglion cells of the toxins produced by mixed infection. According to Deisz,[34] sympathetic atony indicates an unfavorable prognosis.

The shifts in the autonomic status of a tuberculous patient undoubtedly are conditioned by constitutional factors. According to Pende,[35] if the patient exhibits first sympathetic hyperirritability and later parasympathetic hyperirritability a grave prognosis is indicated, whereas, if the patient exhibits primary parasympathetic hyperirritability the disease usually runs a relatively benign course. On the basis of an extensive study of tuberculosis in children, Medowikov and Schenkmann[36] advanced the opinion that the disease usually runs a benign course in children who, as indicated by pharmacodynamic tests, exhibit heightened parasympathetic tonus,

whereas usually it runs a graver course in those who exhibit heightened sympathetic tonus.

Data obtained by animal experimentation support the theory that the character of the local tuberculous lesions and the progress of the infection are influenced by autonomic nerve impulses. In experiments reported by Pigalew and Epstein,[37] rabbits with induced abdominal tuberculous lesions showed increased capacity to combat the disease following bilateral section of the vagus nerves below the diaphragm. In some of the animals the lesions actually underwent regression. On the basis of these results, they advanced the opinion that tuberculous tissue freed from nerve impulses develops increased resistance to infection. The experience of otolaryngologists that cocainization of a tuberculous larynx to relieve pain not infrequently results in regression of the lesions also supports this point of view. In experiments reported by Ponomarew,[38] section of the vagus nerve on the side of the lesion, in rabbits with unilateral pulmonary tuberculosis, retarded the infectious process and tended to limit it to that side.

Histopathological studies of tuberculous lesions in the human body indicate that the tissues react to tuberculous infection according to two modes: (1) they may develop slowly and become quiescent through cicatrization and proliferation; or (2) they may develop rapidly into exudative processes involving dissolution of the tissues. These modes undoubtedly depend on differences in the irritability of the tissues. Low tissue irritability favors cicatrization and proliferation, whereas high tissue irritability favors the exudative process. Tissue irritability is determined in a large measure by the prevailing autonomic status.

The local reactions of the blood vessels undoubtedly constitute one of the most important factors in the progress of a tuberculous lesion. The toxin released at a focus of tuberculous infection causes local vasodilatation which, due to failure of normal reversal to take place, becomes more or less permanent. The focal reactions, therefore, are closely related to the increased permeability of the dilated capillaries. The frequent coincidence of activation of tuberculosis with biological processes such as menstruation, physiological seasonal changes, etc., undoubtedly can be explained on the same basis, since these processes are associated with increased capillary permeability and a shift in the autonomic functional balance. Since clinically advancing tuberculosis is associated with increased capillary permeability and healed tuberculosis with decreased capillary permeability, it may be assumed that increased capillary permeability, regardless of the manner in which it is produced, must influence tuberculous lesions unfavorably, whereas diminished capillary permeability favors improvement. In view of the significant

role of the vasomotor nerves in the regulation of capillary permeability the importance of clinical measures designed to restore the autonomic functional balance in tuberculous disease is indicated.

The passage of air through the respiratory tubes in inspiration and expiration may be hampered either by thickening of the bronchial mucous membrane or by contraction of the bronchial musculature. The effectiveness of sympatheticomimetic agents such as adrenin in reducing edema of the bronchial mucosa undoubtedly depends on their vasoconstrictor action. Spastic contraction of the bronchial musculature involves neuromuscular mechanisms which normally play a significant role in the defense reactions of the upper respiratory tract. The parasympathetic nerves in question may be activated reflexly from the bronchial mucosa by impulses conducted centralward through afferent vagus components, or from other parts of the body by impulses conducted centralward through either visceral or somatic afferent nerves. They may also be activated by impulses emanating from central autonomic centers, particularly parasympathetic centers in the hypothalamus. Asthmatic attacks associated with emotional states can be explained most satisfactorily on the assumption of hypothalamic stimulation.

Bronchial asthma, regardless of its immediate cause, probably always is associated with parasympathetic hypertonus; consequently, blocking or section of the sympathetic nerves in the treatment of bronchial asthma cannot be regarded as a rational procedure. Relief of asthmatic attacks following these procedures, nevertheless, have been reported in many cases, but also a high percentage of failures. The significance of hyperexcitability of the vagus reflex arcs in certain cases of intractable asthma, without recognizable etiological factors, is indicated by the beneficial effects of repeated bronchial relaxation brought about by means of sympathetic stimulants (Barach[39]). The relief in these cases can be explained most satisfactorily on the assumption that a vicious cycle of bronchial spasm has been overcome by the repeated relaxation of the bronchial musculature.

In experiments reported by Braeucker and Kümmell,[40] bronchial spasms simulating asthmatic attacks in man were brought about in apes and rabbits by stimulation of the vagus or sympathetic nerves or the medulla oblongata. Sympathetic stimulation did not produce such spasms following section of the pulmonary branches of the vagi. These results support the assumption that bronchial spasm elicited by sympathetic stimulation involves excitation of the vagus center in the medulla oblongata by afferent impulses conducted centralward by visceral afferent fibers associated with the sympathetic nerves which supply the bronchi. It seems not improbable, therefore, that the relief of asthmatic attacks in certain

cases following section or blocking of the sympathetic nerves to the lungs may have been due to interruption of reflex arcs consisting of afferent spinal nerve components associated with the thoracic sympathetic nerves, ascending neurons in the spinal cord and brain stem and efferent parasympathetic neuron chains, including preganglionic vagus components.

The innervation of the pulmonary arteries and veins is essentially similar to that of the bronchial vessels but less abundant. In cases of hemoptysis involving only pulmonary vessels, measures which cause constriction of the systemic vessels, e.g., the administration of adrenin, may aggravate the bleeding by forcing blood from the bronchial into the pulmonary vessels. Measures which bring about vasodilatation, e.g., the administration of nitrites, tend to relieve engorgement of the lungs by diverting blood into the systemic vessels. Bleeding due to necrosis of lung tissue may involve both pulmonary and bronchial vessels. In such cases also vasoconstriction brought about by styptic agents may be harmful due to the general rise in blood pressure and consequent turgescence of the lungs which tends to cause hemorrhage at other weak points. Measures which produce widespread vasodilatation, on the other hand, may be beneficial due to the diversion of blood from the lungs, which tends to counterbalance the risk of reopening bleeding points. It also facilitates the sealing of bleeding points by means of blood clots.

CONCLUSIONS

The thoracic viscera are innervated through both the sympathetic and parasympathetic divisions of the autonomic nervous system. They are also abundantly supplied with afferent nerve fibers. The extrinsic nerves of the heart and lungs enter the cardiac and pulmonary plexuses. The ganglion cells in these plexuses are connected with the central nervous system through preganglionic components of the vagi; consequently, they are parasympathetic. The sympathetic fibers supplying the thoracic viscera arise in sympathetic trunk ganglia. The sympathetic innervation of the heart comprises both cervical and thoracic sympathetic nerves. Postganglionic fibers arising in all the upper thoracic sympathetic trunk ganglia above the sixth or fifth thoracic segments enter the cardiac plexus. The thoracic cardiac nerves also include afferent components.

With respect to the heart, sympathetic impulses exert an excitatory influence; parasympathetic impulses, an inhibitory influence. Cardiac activity may be influenced reflexly by afferent impulses arising in any part of the body, but those arising in the

carotid sinuses and the cardio-aortic pressoreceptive and chemoreceptive zones are of major importance. Contrary to the theory that constriction of the coronary vessels is mediated through the parasympathetic nerves and dilatation of these vessels through the sympathetic nerves, which has been widely current, most of the more recent findings regarding regulation of the coronary blood flow support the assumption that the parasympathetic innervation of the coronary vessels includes only cholinergic vasodilator fibers, whereas their sympathetic innervation includes both adrenergic vasoconstrictor and cholinergic vasodilator fibers.

With respect to the bronchial musculature, parasympathetic stimulation, in general, elicits contraction; sympathetic stimulation, inhibition. The sympathetic innervation probably includes some cholinergic bronchoconstrictor fibers, at least in some animals. The sympathetic innervation of the pulmonary vessels includes the vasoconstrictor fibers and some vasodilator fibers. The parasympathetic innervation also includes vasodilator fibers. In general, the effect of the vasoconstrictors is more marked than that of the vasodilators.

The weight of experimental evidence favors the assumption that the respiratory center is stimulated mainly through chemoreceptive mechanisms, particularly those associated with the carotid and aortic bodies. Direct chemical stimulation of the respiratory center is not precluded, but this center probably is less sensitive than the peripheral chemoreceptors to some of the normal chemical stimulants.

The prevalence of cardiac pain on the left side seems to be related to the fact that the required volume flow of blood in the left coronary arteries is greater than that in the right. Coronary thrombosis, furthermore, occurs more frequently on the left side than on the right. Impulses of pain of cardiac origin are conducted centralward mainly by afferent components of the thoracic spinal nerves which reach the heart via the sympathetic cardiac nerves.

Pulmonary disease not uncommonly is accompanied by changes in the functional autonomic balance. The changes in the autonomic status observed in tuberculous patients probably are conditioned by constitutional factors. The tissues of the human body react to tuberculous infection according to two modes: (1) the lesions may develops slowly and become quiescent through cicatrization and proliferation; or (2) they may develop rapidly into exudative processes involving dissolution of the tissues. These modes undoubtedly depend on the irritability of the tissues, which is determined in a large measure by the prevailing autonomic status. Clinically advancing tuberculosis commonly is associated with increased cap-

illary permeability; healed tuberculosis with decreased capillary permeability. Since capillary permeability is regulated through the vasomotor nerves, the importance of therapeutic measures designed to restore the autonomic functional balance in tuberculous patients is indicated.

Bronchial asthma probably always is associated with parasympathetic hypertonus. Therapeutic measures for the relief of bronchial asthma which involve blocking or interruption of sympathetic nerves, therefore, are irrational.

CONCLUSIONES

Las vísceras torácicas reciben su inervación por medio de las dos divisiones del sistema nervioso autonómico: la división simpática y la parasimpática. También están provistas abundantemente de fibras nerviosas aferentes. Los nervios extrínsecos del corazón y los pulmones entran a los plexos cardíaco y pulmonares. Las células ganglionares de estos plexos están unidas al sistema nervioso central por medio de componentes preganglionares de los nervios vagos y son, por consiguiente, parasimpáticas. Las fibras nerviosas simpáticas que suplen las vísceras torácicas originan en ganglios del tronco simpático. La inervación simpática del corazón comprende nervios simpáticos cervicales y torácicos. Fibras postganglionares que originan en todos los ganglios torácicos superiores del tronco simpático, situados arriba del quinto o sexto segmento torácico, entran al plexo cardíaco. Los nervios cardíacos torácicos también contienen componentes aferentes.

Los impulsos simpáticos ejercen una influencia excitativa en el corazón y los impulsos parasimpáticos una influencia inhibitoria. La acción cardíaca puede ser influenciada en forma refleja por medio de impulsos aferentes que originen en cualquier parte del cuerpo, pero los que originan en los senos carótidos y en las zonas presoreceptivas y quimioreceptivas cardio-aórticas son de mayor importancia. Contrario a la teoría, que ha sido generalmente aceptada, de que la constricción de los vasos coronarios se lleva a cabo mediante los nervios parasimpáticos y la dilatación de estos vasos mediante nervios simpáticos, la mayor parte de los descubrimientos más recientes, tocantes a la regulación de la circulación coronaria, confirma la hipótesis de que la inervación parasimpática de los vasos coronarios contiene sólamente fibras vasodilatadores colinérgicas, mientras que la inervación simpática contiene fibras vasoconstrictoras adrenérgicas así como fibras vasodilatadoras colinérgicas.

Con respecto a la musculatura bronquial, la estimulación parasimpática, en general, provoca contracción y la estimulación simpática inhibición. La inervación simpática probablemente contiene

algunas fibras broncoconstrictoras colinérgicas, por lo menos en algunos animales. La inervación simpática de los vasos pulmonares contiene fibras vasoconstrictoras y algunas fibras vasodilatadoras. La inervación parasimpática también contiene fibras vasodilatadoras. En general, el efecto de las vasoconstrictoras es más marcado que el de las vasodilatadoras.

La mayor parte de la evidencia experimental favorece la hipótesis de que el centro respiratorio es estimulado principalmente por medio de mecanismos quimioreceptivos, particularmente los que están asociados a los cuerpos carótidos y aórtico. Esto no excluye la posibilidad de la estimulación química directa del centro respiratorio, pero este centro es probablemente menos sensible a algunos de los estímulos químicos que los quimioreceptores periféricos. La frecuencia del dolor cardíaco en el lado izquierdo parece tener relación con el hecho de que el volumen circulatorio requerido en las arterias coronarias izquierdas es mayor que en el lado derecho. Además, la trombosis coronaria es más frecuente en el lado izquierdo que en el derecho. Los impulsos de dolor de origen cardíaco son conducidos al sistema nervioso central principalmente por medio de componentes aferentes de los nervios espinales torácicos que llegan al corazón por vía de los nervios cardíacos simpáticos.

No es raro que la neumopatía esté acompañada de alteraciones en el equilibrio funcional autonómico. Las variaciones en el estado autonómico que se observan en pacientes tuberculosos probablemente dependen de factores constitucionales. Los tejidos del cuerpo humano reaccionan a la infección tuberculosa de dos modos: 1) Las lesiones pueden desarrollarse lentamente y volverse inactivas debido a cicatrización y proliferación, o 2) pueden desarrollarse rápidamente en forma de procesos exudativos que causan la disolución de los tejidos. Es indudable que el modo depende de la irritabilidad de los tejidos, lo cual lo determina en gran parte el estado autonómico prevalente. Tuberculosis que está avanzando, desde el punto de vista clínico, viene acompañada frecuentemente de una aumento en la permeabilidad de los vasos capilares; tuberculosis cicatrizada, de una dismunución en la permeabilidad de los vasos capilares. En vista de que la permeabilidad de los vasos capilares está regulada por nervios vasomotores, es fácil darse cuenta de la importancia de terapéuticas cuyo propósito es el restablecer el equilibrio funcional autonómico en pacientes tuberculosos.

Probablemente el asma bronquial está acompañada siempre de un hipertono parasimpático. Por consiguiente, terapéuticas para el alivio del asma bronquial que incluyen bloqueo o interrupción de los nervios simpáticos son ilógicas.

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Atypical Pneumonia of Unknown Etiology

A Clinical, Roentgenological, and Pathological Correlation

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INTRODUCTION

Terminology and Definition—Considerable interest has been aroused during the past few years in a syndrome variously designated as Primary Atypical Pneumonia, Non-Bacterial Bronchopneumonia, Virus Pneumonia or Pneumonitis, Interstitial Pneumonia, Interstitial Bronchopneumonia, and so forth. While its causative agent or agents are unknown, its kinship, clinically, roentgenologically and pathologically with the group known to be virus in origin, such as Influenza A or B, Ornithosis (Psittacosis), and others, justifies our speaking of it as a virus disease. Hence it would seem advisable to use the name Atypical Pneumonia in a generic sense; in one group naming the causative virus and in the other, stating the etiology is unknown.

Current Misconceptions—A prevalent concept among writers is to consider it a disease entity, whereas it should be considered one manifestation of a general disease as expressed by Reimann[1] and concurred in by Francis.[2] The latter states that the pneumonia may well be a physiological accident, implying, thereby, that the disease is one of the upper respiratory tract that has gravitated to the lungs.

Nor should it be considered a new disease, having been found in sections of lungs removed from soldiers during the Civil War (MacCallum) and preserved in the Army Medical Museum. The pathology in our one case resembles that seen by us in the influenzal and streptococcal pneumonias and empyemas occurring in military camps in 1918.[3,4] In civilian practice for many years we have observed sporadic cases difficult to differentiate from those observed in the last war and in this one.

Another error is the frequency with which atypical pneumonia, modified or changed by the invasion of secondary bacterial pathogens is confused with the disease in its pure or uncomplicated form (Fig. 5).

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The accompanying photomicrographs are from a patient who entered the hospital with an atypical pneumonia. He was discharged following clinical recovery. After two weeks he was readmitted to the hospital with a recurrence of his atypical pneumonia complicated by an acute glomerulonephritis. He died two weeks later of his nephritis and a bacterial (pneumococcus) pneumonia superimposed upon his atypical pneumonia.

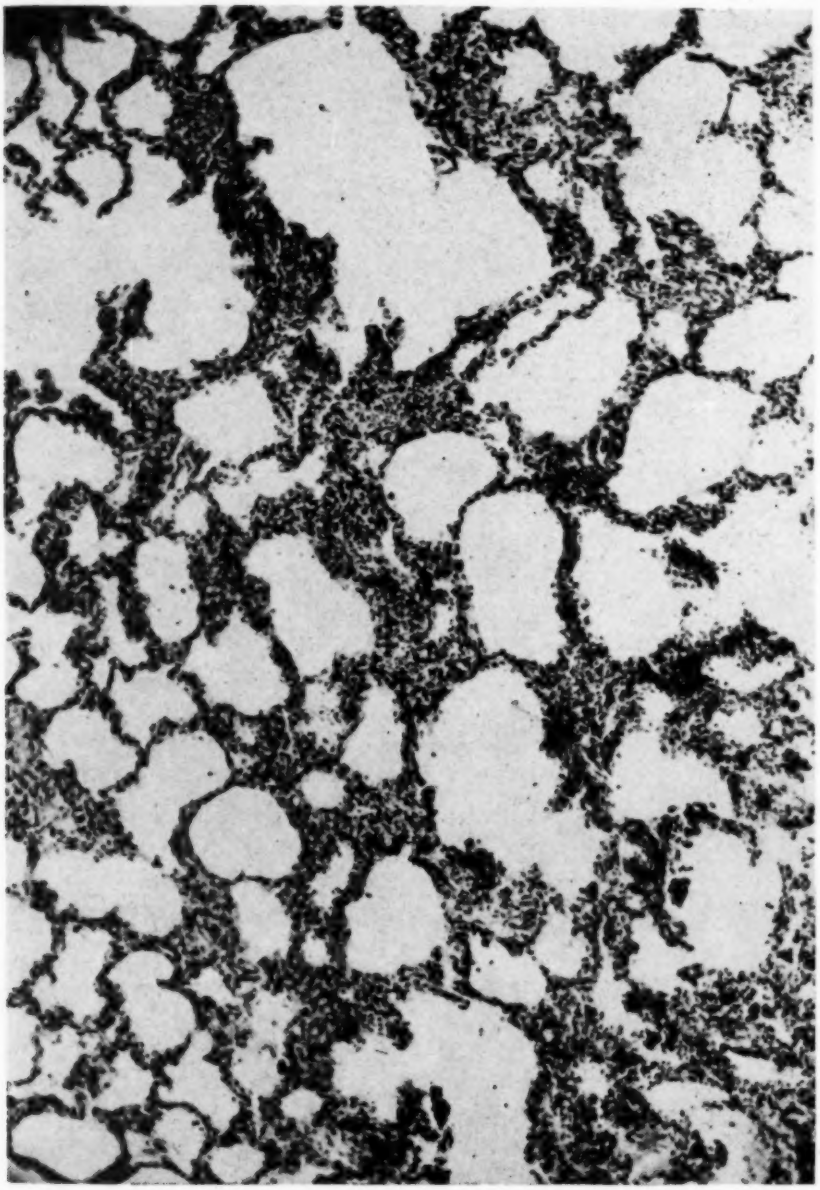


Fig. 1.—Magnification 120x. The interstitial tissue composing the interalveolar septa is oedematous, shows lymphangiectasia and is infiltrated by mononuclear cells, a scattering of histiocytes, many plasma cells and a few polymorphonuclear leukocytes and red cells. The lining of the alveoli for the most part retains its continuity but in many places is ruptured. The endothelial cells are swollen and show a tendency towards a cuboidal form. Notable is the absence of any exudate within the alveoli many of which are dilated.

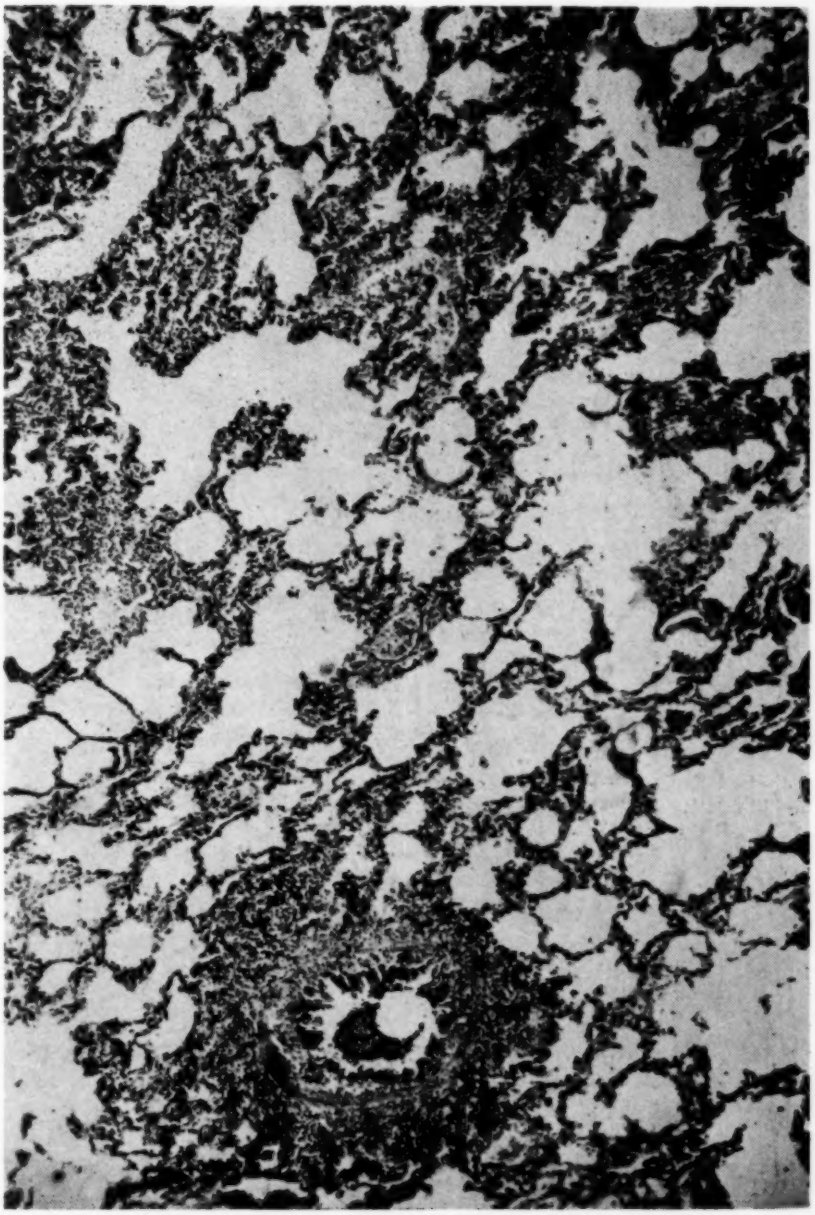


Fig. 2—Magnification 80x. In this section, the "collar" encircling the bronchus represents the supporting tissue infiltrated with mononuclear elements, collapsed alveoli and some alveoli filled with a serous and mononuclear cellular exudate. Emphysema due to rupture of septal walls and dilatation of alveoli with areas of atelectasis is in evidence. For the most part the alveoli are free of exudate although a few show a serous and cellular exudate composed of mononuclear cells. The muscular layers of the bronchi are shredded, the bronchial wall itself is markedly ŏedematous and infiltrated with cells almost all being mononuclear cells. The mucosa is fragmented, ulcerated and contains a plug largely composed, in this instance, of polymorphonuclear leukocytes. Mononuclear cell plugs are common.

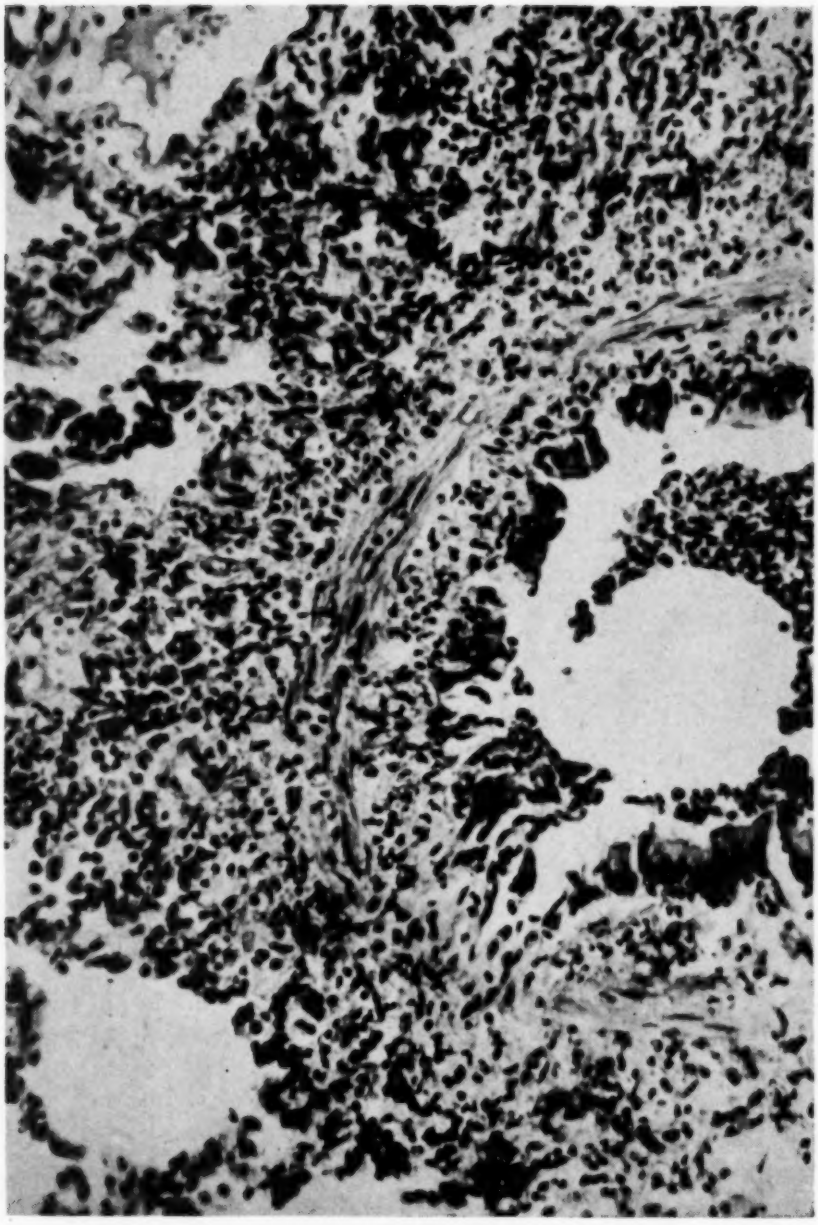


Fig. 3—Magnification 325x. A section of Fig. 2 under higher power.

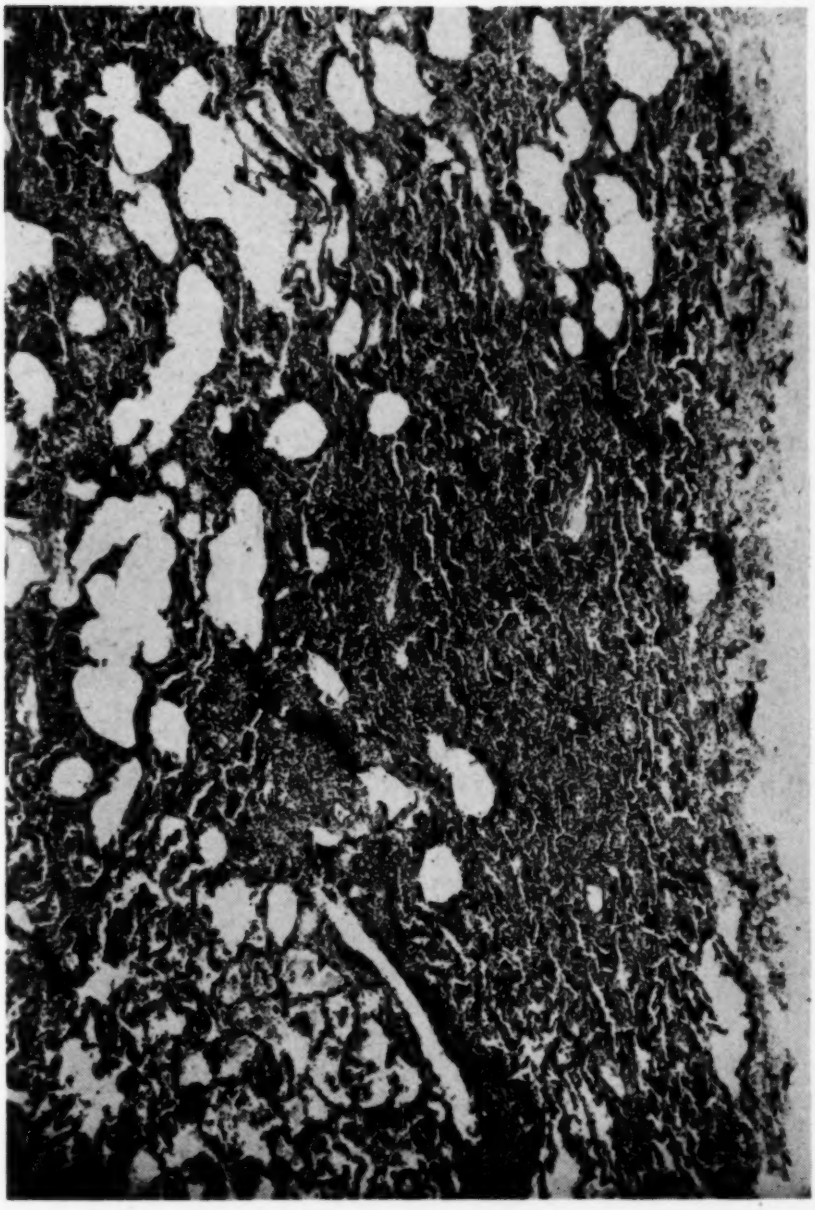


Fig. 4—Magnification 60x. Adjacent to the bronchus (cut tangentially) with its marked peribronchial thickening there is a mass of atelectatic alveoli, while on the opposite side some alveoli show an exudate composed of serum and some mononuclear cells. Such when present is the characteristic exudate of interstitial pneumonia.

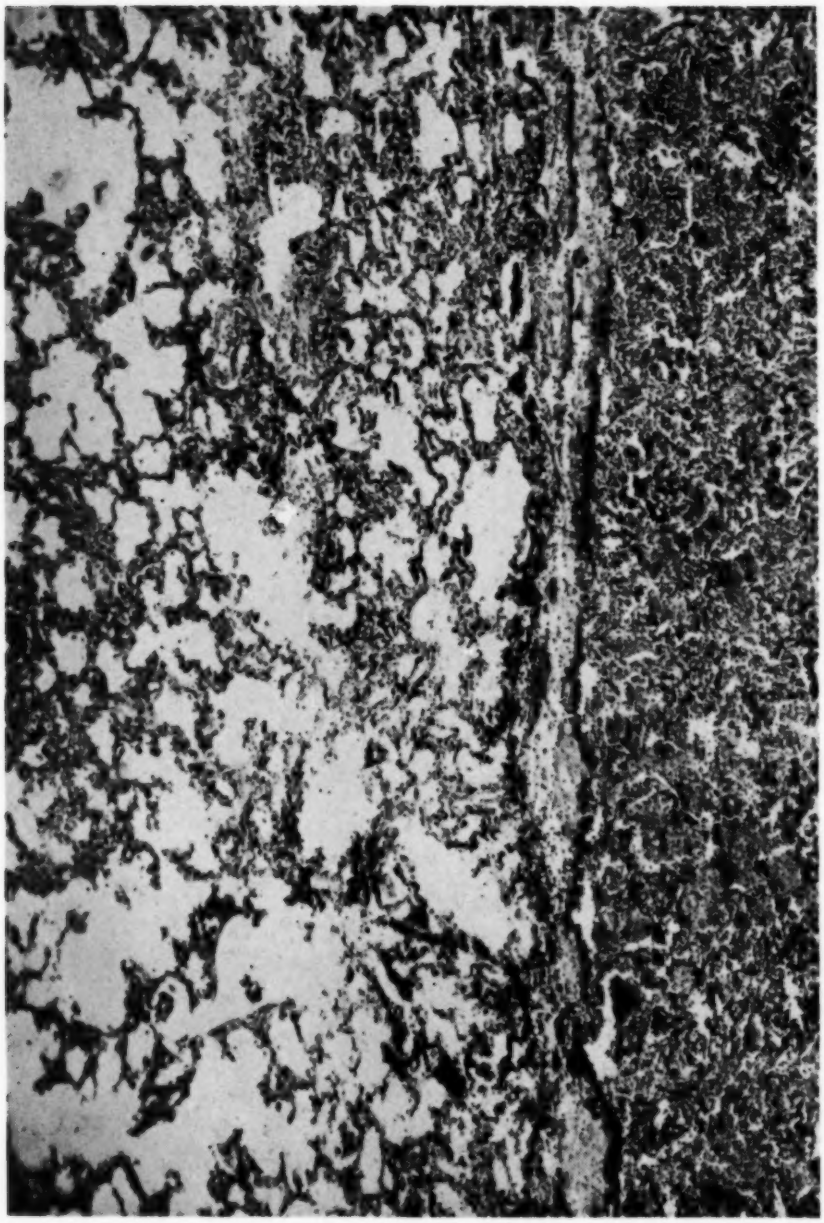


Fig. 5—Magnification 80x. On the one side of the interlobular septum is an area of typical interstitial pneumonia, showing emphysema, atelectasis and bronchioles filled with a mononuclear cellular exudate. On the other side when view under high power an exudative bacterial pneumonia can be recognized. The alveoli throughout are filled, however, with an exudate composed almost entirely of polymorphonuclear leukocytes, serum, fibrin and red cells, in which pneumococci can be identified. This portion of the section represents a confluent bacterial lobular pneumonia superimposed on an interstitial pneumonia.

Early recognizing this last misconception we felt it necessary to set up certain criteria to which the syndrome must conform before a diagnosis of Atypical Pneumonia—Etiology Unknown, may be made.

SOURCE OF OUR MATERIAL

The source of our material consisted of a correlation of the studies made of: (1) the physical and x-ray findings in atypical pneumonia; (2) the pathology, as seen in our own case who died with atypical pneumonia and that submitted to us by the Army Medical Museum; (3) the types of pneumonia we observed in measles and scarlet fever.

The data upon which the impressions in this paper are based were derived from the above studies and a critical analysis of some five hundred patients. These patients represented a cross section of some six thousand cases of acute epidemic respiratory tract infection, which entered the Station Hospital, Fort Custer, Michigan, from December 1, 1942, to June 1, 1943. Studying these five hundred patients in as much detail as circumstances and facilities permitted, we observed that sixty per cent terminated during the period of invasion without involvement of the lungs; that fifteen per cent justified the diagnosis of bronchitis based on physical findings; and that twenty-five per cent had pneumonia proven by x-ray.

CRITERIA USED AS A BASIS FOR DIAGNOSIS

The criteria which we have established as a basis for diagnosis are: The nature of the onset; the physical findings checked with the roentgenograms; the course of the disease; the character of the x-ray shadows; the white cell count; sputa examinations; and the response to the sulfonamides. Fever and types of fever curves present no characteristics peculiar to the syndrome. In reviewing the fever charts one sees that any type of fever curve may be present in any phase of the disease and that fever often bears no relation to the physical or x-ray findings (Figs. 6, 7, 8, 9).

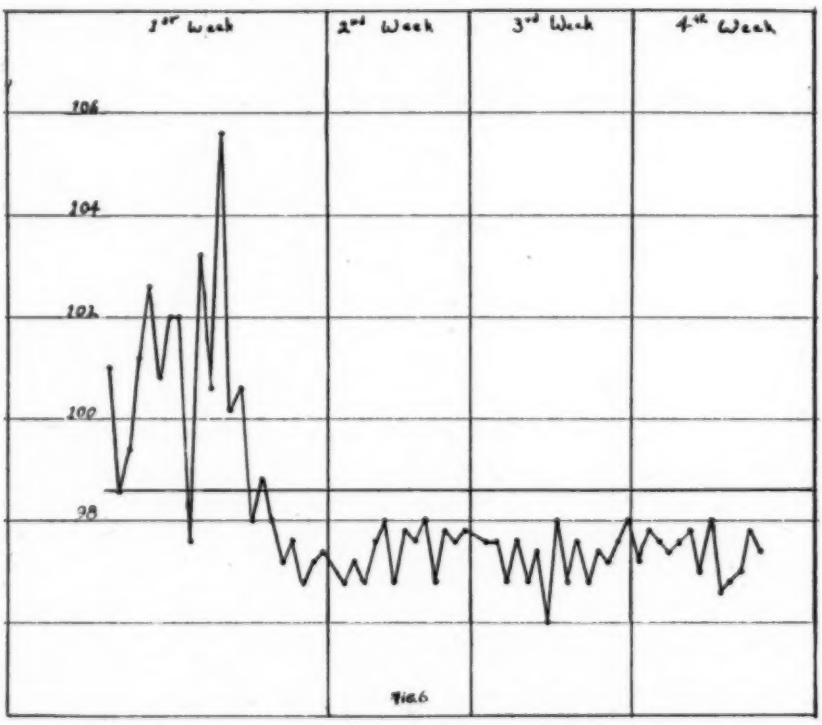
The Onset—This is variable and may be sudden, gradual or even insidious. In most instances the syndrome is ushered in as a wet nasopharyngitis which more or less rapidly extends downward, taking on the character of a laryngo-tracheitis and often a bronchitis before the pneumonia becomes evident. Generally it lacks dramatic suddenness but in almost all cases gives the impression of an acute intoxication, generally of moderate severity.

The Physical Findings—In contrast to the findings elicited in the bacterial types of lobular and lobar pneumonia, one is struck by the number of patients exhibiting physical findings out of proportion to the x-ray evidences and the course of the disease. While these find-

ings depend in considerable measure on the phase of the disease process, they are characterized by elusiveness and difficulty of detection. They depend upon a critical sense of touch to detect areas of impaired resonance and upon an ear attuned to fine shades of difference in tone production and in the perception of adventitious sounds.

The Course—Here again much depends upon the phase of the disease with which we are dealing. A stormy onset may terminate abruptly (Fig. 6); one mild in its invasion period may continue for weeks or months marked by remissions and exacerbations (Fig. 7); or the course throughout may remain subdued with a low grade fever (Fig. 8), slight chills or chilliness, followed by sweats. Or, the patient may go on to clinical recovery, but for weeks or months persistent physical or x-ray findings tell us the process still smoulders. Indeed a sudden alightening frequently occurs. The course is bizarre, confusing, confounding and unpredictable.

The X-ray—"X-ray pneumonia" is a term often applied to the disease because a chance or routine x-ray film may be all that reveals its presence. The x-ray findings of the various phases of the disease generally are distinctive. They are not characteristic. They must be differentiated from the shadows seen in bacterial and other virus pneumonia, from tuberculosis, from bronchiectasis associated with a peribronchial reaction, and so forth. But when corre-



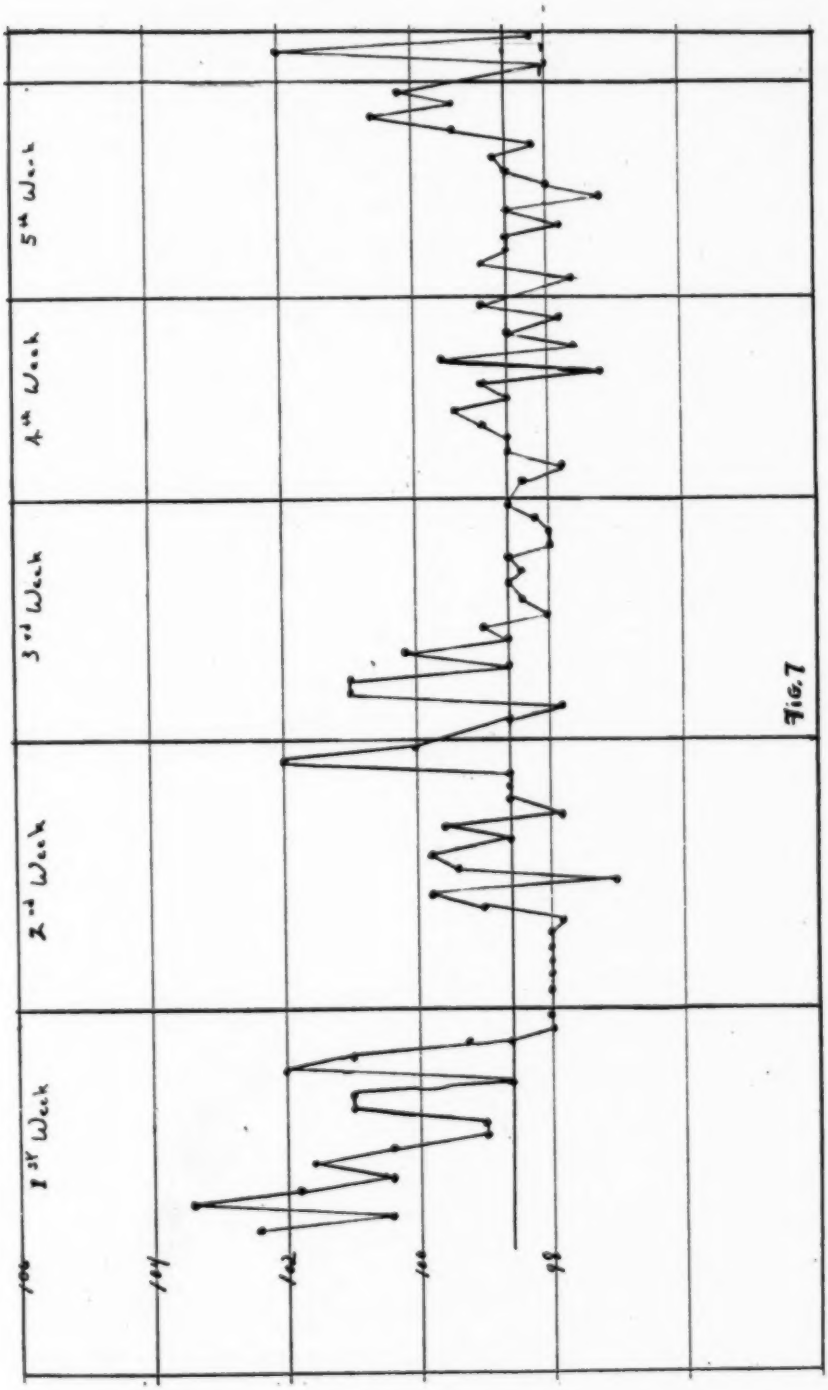


Fig. 7

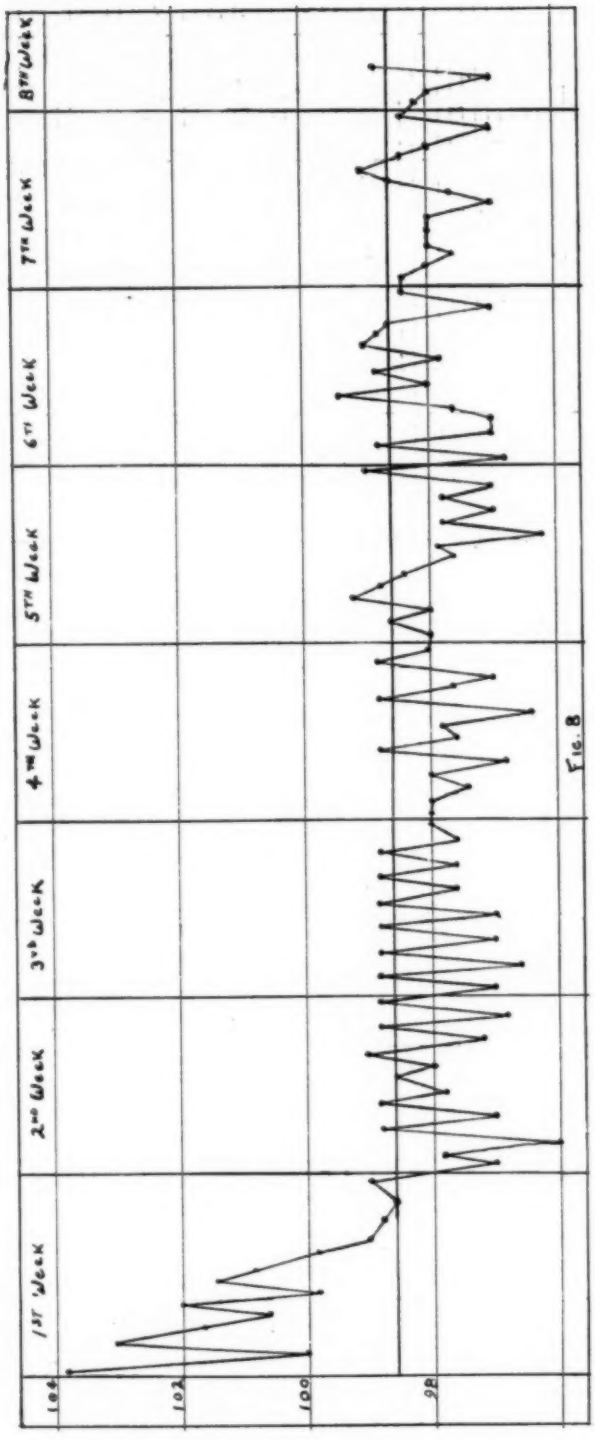

1st Week
2nd Week
3rd Week
4th Week
5th Week
6th Week
7th Week
8th Week
104
102
100
98

FIG. 8

lated with all other data often they may be interpreted as indicative of atypical pneumonia.

The White Cell Count—With few exceptions the leukocyte count is but slightly elevated, normal or low throughout the course of the disease. Often, if the pneumonia lasts a few weeks the count rises to the level of a distinct leukocytosis, later to fall. When the count reaches fourteen, sixteen, or eighteen thousand and when it remains at these heights, generally it indicates the presence of bacterial pathogens.

The Sputa—Early, sputa are absent, later becoming mucoid, expectorated as homogeneous grayish gelatinous plaques, non-frothy, streaked with blood and free of bacteria. Later they may become thinner, whitish and even frothy and abundant, but still remain free of pathogens except in complicated cases.

The Response to Sulfonamides—Enough evidence has accumulated to say that pure atypical pneumonia does not respond to the sulfonamides. However, in those patients running a prolonged course or who have a persistent cough, and in whom the sputa show bacterial pathogens the response to these drugs often is prompt. The cough subsides, bacteria disappear from the sputa, and the leukocyte count falls to normal. Where such response does occur it would seem to indicate the presence of secondary invaders.

PHASES OF THE SYNDROME

The syndrome as we have observed it differs in no wise from that so ably described by Reimann,[1] Finland and Dingle,[5] Kneeland and Smetana,[6] McLeod,[7] Longcope,[8] Moore,[9] Murray,[10] Dingle et al.[11]

When the upper respiratory tract is affected, the condition may be designated as a nasopharyngitis, a laryngitis or a laryngo-tracheitis. So, when the infection extends into the lung we find it takes on certain characteristics depending upon what portion of the pulmonary tract is predominantly affected.

Thus we find on the basis of physical and x-ray examinations and to some extent on the course, the syndrome may be divided into the bronchitic, the peribronchitic, the alveolar and the bronchoalveolar. While any one of these phases may exist independent of the other, more often several phases coexist. Equally often one phase merges into another. Hence it is to be remembered that the terms as used only apply to that part of the pulmonary parenchyma predominantly affected and that any parts may be involved simultaneously or successively.

In Fig. 9 with its accompanying roentgenograms Figs. 10 and 11, we find the completed story of the type of case commonly seen.

Bronchitic Phase—This generally involves the bronchi through-

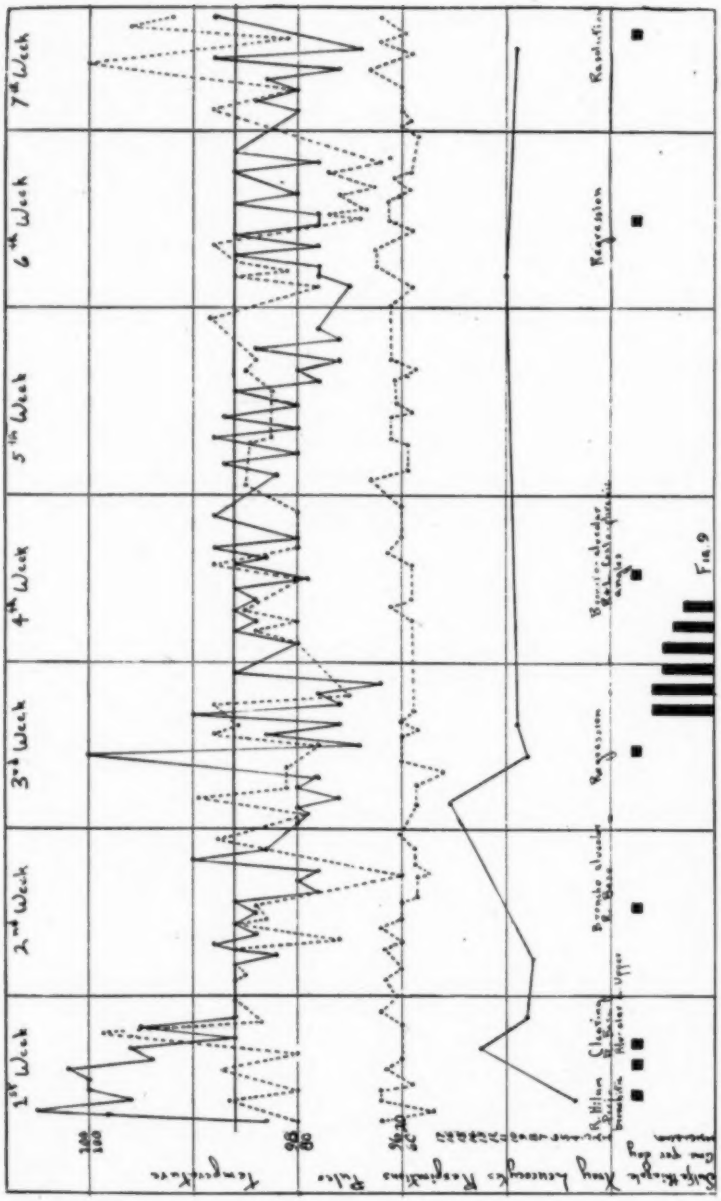


Fig. 9—Patient here represented entered hospital four days after onset of "cold," chilliness, fever, with physical findings of impaired resonance and blowing breath sounds in right base. Roentgenogram on the second day of hospitalization is shown in Fig. 10. Roentgenogram on the fifth day of hospitalization is shown in Fig. 11. Physical examination at this time showed impaired resonance over the left upper lobe anteriorly with fine rales. From then on both on physical and x-ray examinations the lesions began to regress, despite bouts of chilliness and fever. During the fourth week, rales reappeared in the right and left costophrenic angles. A roentgenogram showed new lesions.

out both lungs beginning as a nasopharyngitis and terminating as a mild or severe bronchitis.

The onset in this as in all phases of the syndrome is varied and is frequently preceded for some days or weeks by a wet nasopharyngitis. An abrupt onset characterized by headache, malaise, chilliness or distinct chills, sore throat, fever, and a hacking, nonproductive cough, accompanied by tightness in the chest and general body aching, is equally frequent.

When a patient is stricken thus he takes to bed with a burning fever in which the temperature mounts to 101° F., 102° F., 103° F., and even higher. Under sedation he falls asleep, the fever abates, and he awakens covered with a drenching sweat. The following day or at most two or three, he becomes fever free, the cough subsides, he feels well, and he is returned to duty with a diagnosis of naso-

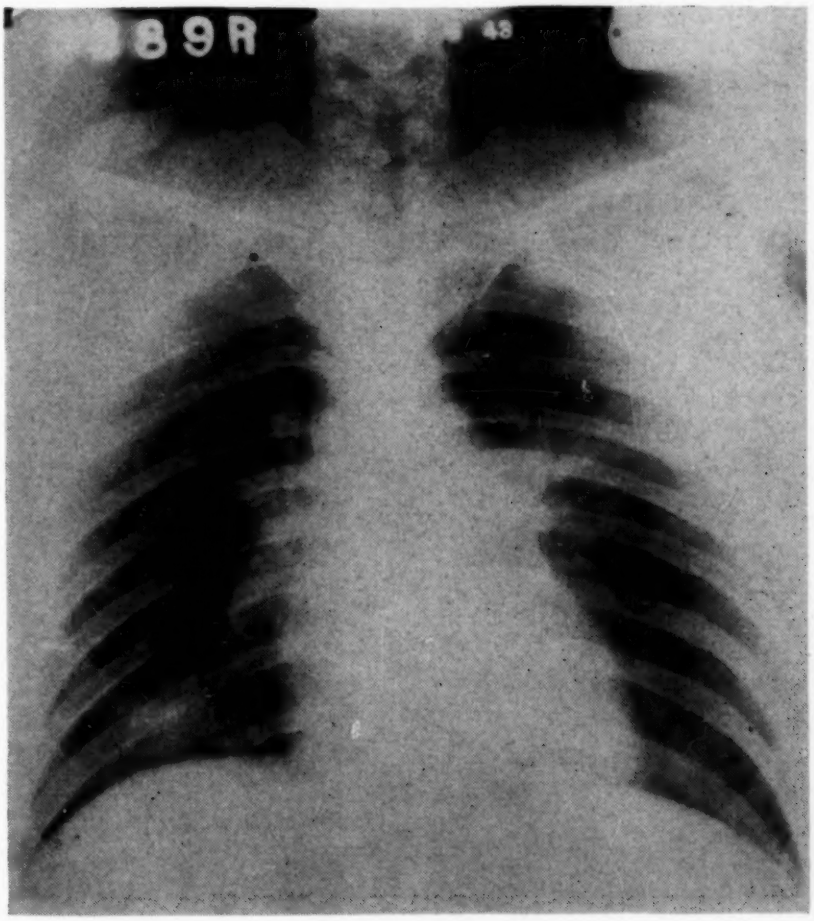


Fig. 10—Shows increased density of both hilar shadows; a patchy density radiating from the left hilus; and soft mottling with feathery margins in the right base.

pharyngitis—cured. He has gone through the nasopharyngeal phase.

Should the illness continue, we find him at the end of three or four days, languid and limp, the result of an overwhelming infection. The fever may persist, though at a low level, or it may subside. He frequently develops hoarseness and has episodes of explosive coughing. The conjunctivae are injected, the pharynx is reddened and may show patches of a gelatinous exudate. The lungs show rales, large squeaky sibilant rales and rhonchi, widely scattered, sometimes localized. At the end of ten days or thereabouts, though a little pale and wan, the soldier is returned to duty having passed through the bronchitic phase of the disease.

Let the illness continue from here on or let it start with a history of well-being until its inception or a history of several attacks of mild colds throughout the season. Here we find the patient hard hit, early developing cyanosis and dyspnoea, coughing up plaques of sputum or having no sputa at all. Through the days and weeks he

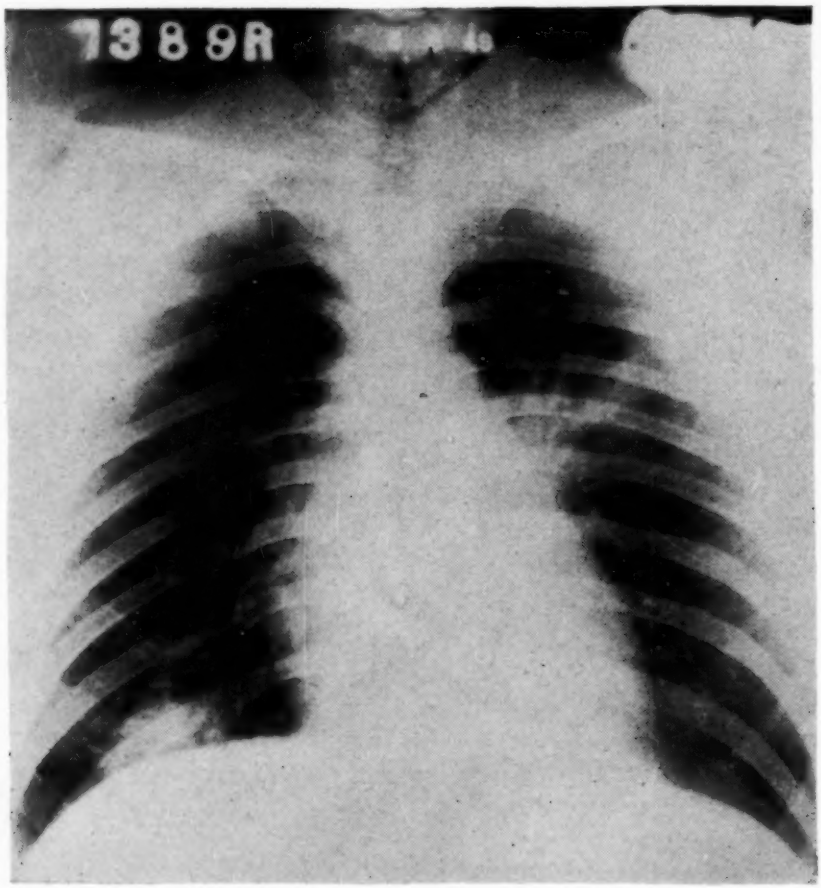


Fig. 11—Shows outward progression of the lesion in the left hilus; and early regression in the right base.

suffers repeated attacks of chilliness with rises of temperature followed by sweats or he may have little or no fever. The cough persists, not so severe or racking; his muscles and joints feel sore and ache but are not tender. He breathes with his mouth agape; his respirations continue a bit rapid; his pulse is full but does not correspond in rate to his temperature. His leukocytes have risen from the leukopenic or normal level to ten or twelve thousand. Rales, sibilant rales, and occasionally a patch of transitory finer rales here and there preceded by a questionable suppression of the normal vesicular murmur—one thinks of atelectasis—but no pneumonia.

The roentgenologist is mustered into service. Day by day he, too, watches and feels amply rewarded if confidently he can say bronchitis. And yet the patient goes on with bouts of chilliness, fever and sweats, ill feeling, remissions, exacerbations, reinfections (Fig. 7). Then after weeks or months—in one of our cases six months—he is discharged as a bronchitis and is said to have been in the bronchitic phase.

Though clinically accurate, bronchitis as a diagnosis leaves much to explain. When one reviews the pathology as seen in Fig. 1 and notes the fixation of the alveoli resulting from the infiltration of the septa by mononuclear cells and edema, one cannot refrain from the impulse of offering this as an explanation for the severe bronchitis and of calling the bronchitis in reality an interstitial pneumonia.

Peribronchitic Phase—Here is something the presence of which we feel we can prove. Rewarded by the discovery of something tangible we forget how or when it began; we remember only that its onset and course did not differ from the other phases.

We examine the chest. There is little to observe on inspection, not even dyspnoea. On percussion over the base or bases the resonance is or seems to be impaired. We listen. Few or many localized or scattered rales, sibilant, coarse, squeaking rales. The breath sounds seem to be suppressed, the soft rustle of opening air sacs is gone and there is a gentle blow with prolongation of the expiratory phase.

The roentgenologist presenting his film points out the increased size of the hilar shadows and the increased density of the trunkal markings radiating as shafts towards the diaphragm (Fig. 12). This is the distinctive finding roentgenologically in the peribronchitic phase. Yet one must be careful lest it represent an old bronchitis or bronchiectasis.

Not infrequently by the time these findings present themselves the patient is free of fever and well on the road to recovery, although the x-ray evidences of residua may persist for many days or weeks or occasionally even months.

In Figs. 2 and 3 we find the pathological reason for these findings.

The bronchial walls are markedly thickened and the peribronchial alveoli atelectatic or filled with exudate massed about and encircling the bronchi. This may account for the suppressed alveolar murmur and the bronchial quality of the breath sounds.

Alveolar Phase—Concurrently with, rarely independent of, sometimes following, the bronchitic or peribronchitic phase, the alveolar phase makes its appearance.

What is this alveolar phase? It is represented roentgenologically by (a) the small irregular, slightly opaque shadow or shadows lurking among the thickened bronchi (Fig. 13); (b) that well-circum-

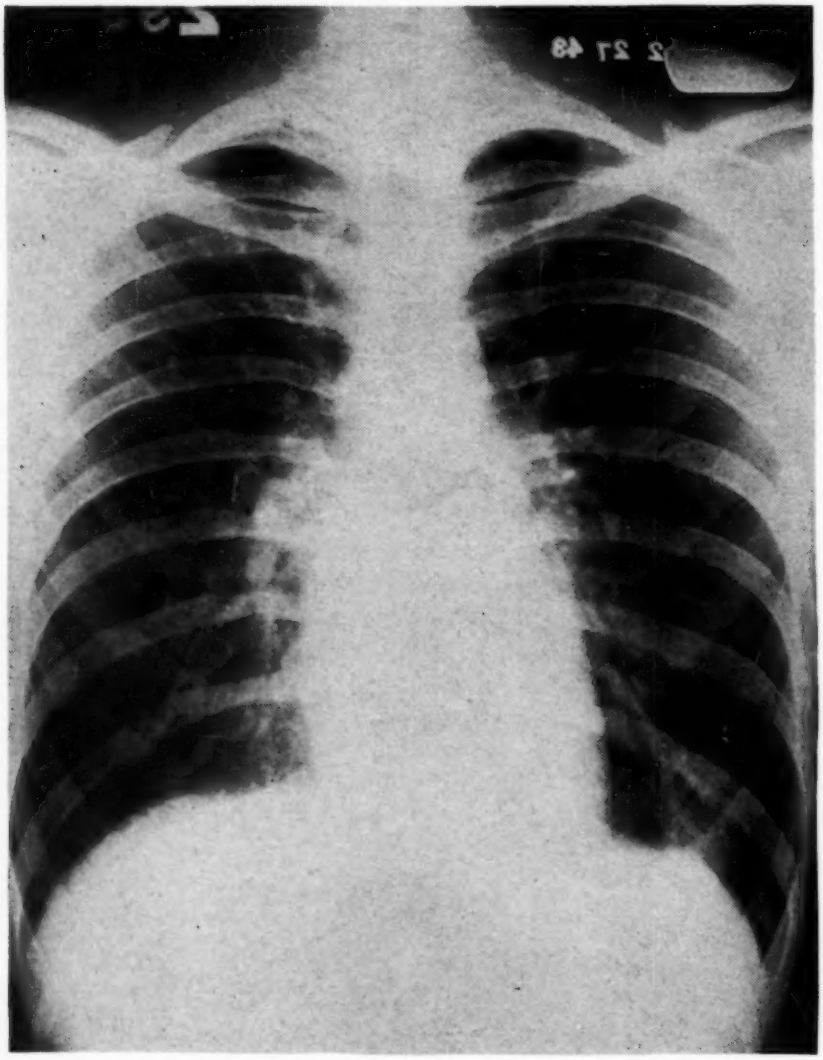


Fig. 12—*Peribronchitic Phase*, showing increased hilar shadows and trunkal markings bilaterally; also a shadow of soft density adjacent to the bronchi in the left base.

scribed or ill-defined shadow, soft, fleecy, ground-glass in appearance which spreads out fan-like from the hilus (Fig. 13), or which hangs unsuspended here and there throughout the lung fields (Fig. 11); (c) that extensive lesion which, occupying the greater part of a lobe, reaches out toward the periphery or the interlobar pleurae (Fig. 14).

It is that mystifying lesion, typical of atypical pneumonia, but equally suggestive of many other conditions as for example, tuberculosis—that lesion which is often confused with the bacterial pneumonias (Figs. 5, 10, 14). Or it may not be a pneumonia at all but an area of atelectasis (Fig. 4) resulting from bronchial occlusion (Figs. 2, 3). Perhaps it represents alveoli filled with a serous exudate, or merely an area of congestion.

It is this phase which so often has been spoken of as "x-ray pneumonia" because of the absence or paucity of physical findings. While it generally appears at the onset of the disease or shortly thereafter, its appearance may be delayed until the patient is clini-

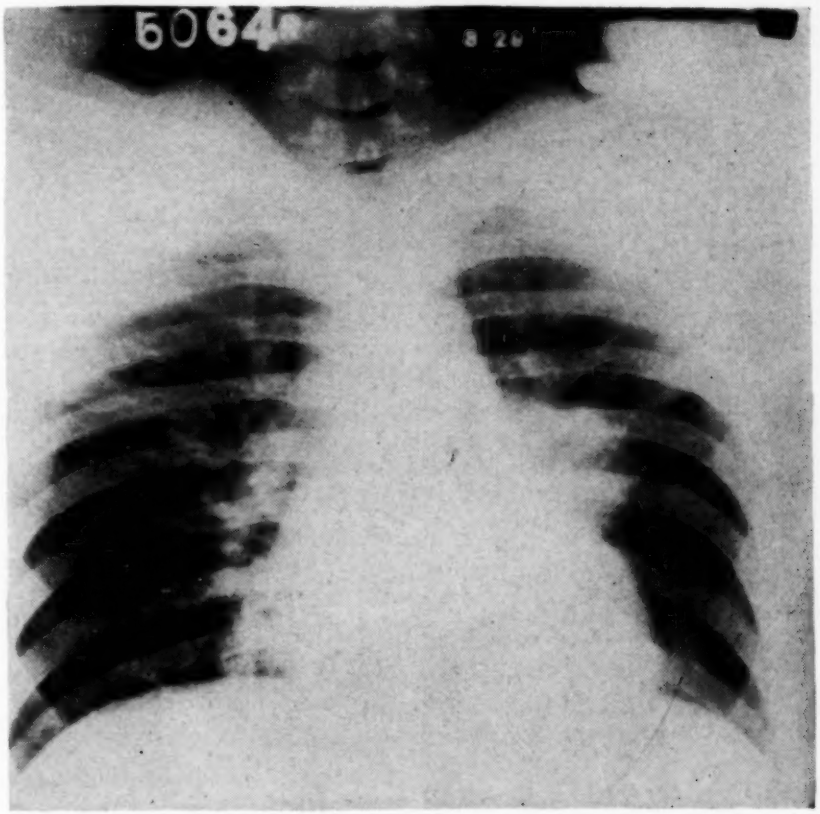


Fig. 13—*Alveolar Phase*, showing irregular soft densities in the lung parenchyma at the right base and in the cardiophrenic sinus; also ill-defined density radiating from the left hilus.

cally well. It may last a few days or even weeks but generally disappears not later than the fourteenth day. Often it reappears in some other part of the lung field.

Chilliness and fever generally usher in its appearance. Bouts of chilliness and fever mark its course. Often it appears unannounced, a chance or routine film revealing its presence. Often there is no fever or the rise in temperature bears no relation to the appearance of the shadow (Fig. 9).

It is in this phase that on physical examination one is confronted with the difficulty of perceiving lessened mobility, so constant in lobar (pneumococcus) pneumonia; of detecting slight degrees of impaired resonance; of determining whether the breath sounds are suppressed or the vesicular murmur is absent or of recognizing an increased transmission of the whispered voice. The fine rales characteristic of the invasion period of bacterial pneumonia are almost

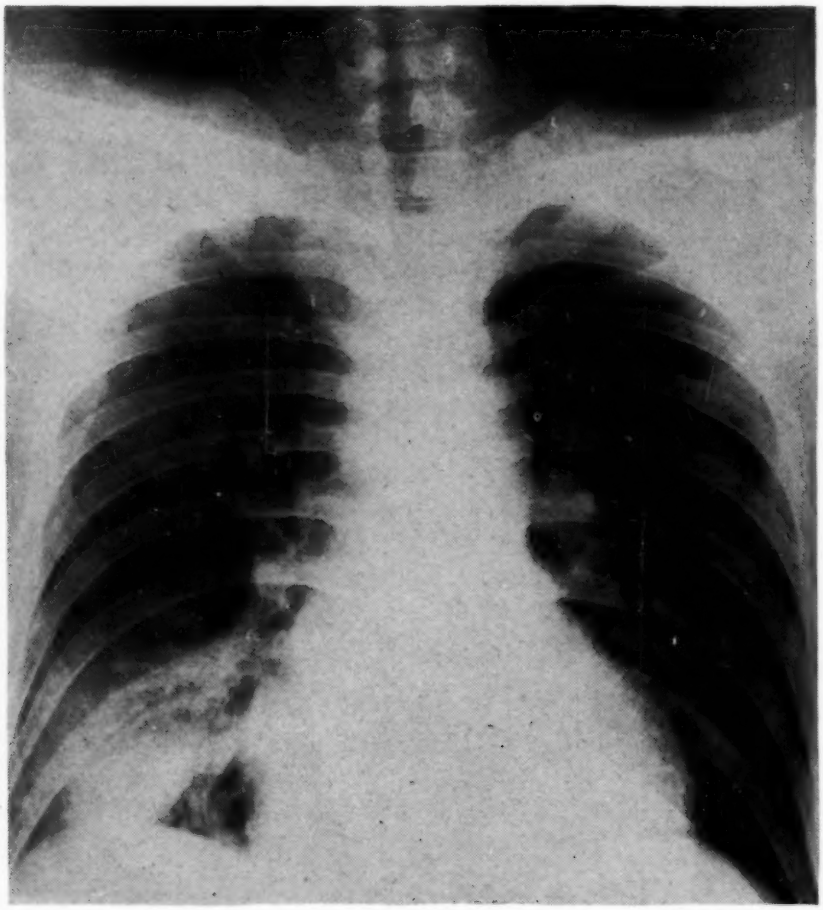


Fig. 14—*Alveolar Phase*, showing fan-shaped shadow of soft density, resembling lobar pneumonia.

always absent and may never appear even in the stage of regression.

At this juncture it seems opportune to say a word about what one may speak of as a lobar phase. This lobar phase, affecting the greater part of a lobe or even an entire lobe, rightfully should be considered nothing more than an extensive pneumonia, alveolar in type.

This lesion both upon physical and x-ray examination—except for the lessened density of the shadow—is indistinguishable from lobar pneumonia bacterial in origin. Even in this lobar phase the leukocyte count is normal or but slightly elevated, the sputa are negative for organisms and the patients appear less ill than those suffering from bacterial pneumonia. The course may be relatively short or it may be prolonged and there is no response to the sulfonamides.

On the other hand we have encountered a few such patients who, following the use of sulfonamides, had an abrupt fall of temperature going on to complete recovery. Opinion on this phase must be reserved.

Broncho-Alveolar Phase—As the name implies, we find in this phase an involvement of both the bronchi and the alveoli (Fig. 15). On physical examination we may find patchy areas of dullness or impaired resonance over which there may be vesiculo-bronchial breathing or merely a prolongation of the expiratory phase of respiration. Here the breath sounds may be suppressed or the normal vesicular murmur absent. Some large bronchial rales generally are present throughout as well as scattered bursts of fine vesicular or bronchiolar rales.

Almost frequent enough to be considered peculiar to this phase of the pneumonia, is a phenomenon in which over small areas there is a suppression or absence of the vesicular murmur during ordinary respiration. Forced inspiration results in a sudden explosive "puff," followed by a burst of fine moist rales and the reappearance of the normal alveolar rustle. This phenomenon tends to repeat itself during the examination and probably is representative of atelectasis.

On x-ray examination this phase shows itself as a diffuse mottling (Fig. 15) or as small, denser, tapioca-like granules, confined to the bases, adjacent to the increased trunkal markings or scattered throughout the parenchyma (Fig. 15). Both types frequently coexist. It is conceivable that these lesions in many instances represent atelectasis or areas of congestion.

Many patients in whom this phase is or becomes predominant, frequently undergo a prolonged course marked by an irregular type of fever (Fig. 7) or little or no fever (Fig. 8). These patients show migrating or reinfectious types of lesions, detectable sometimes by

physical signs, sometimes by x-ray, sometimes by both, and occasionally by neither.

Frequently in this phase a superimposed bacterial infection occurs in which the sputa become laden with pathogenic organisms. Often an intercurrent infection such as tonsilitis or sinusitis develops. In such cases the white count invariably rises to fourteen, sixteen, or eighteen thousand. It is in these secondarily infected and complicated cases that we see a response to the sulfonamides.

COMPLICATIONS

Is pleurisy, serous, fibrinous, purulent or fibrous, occurring with or following upon atypical pneumonia one of the manifestations of atypical pneumonia? In about one half of our patients in whom

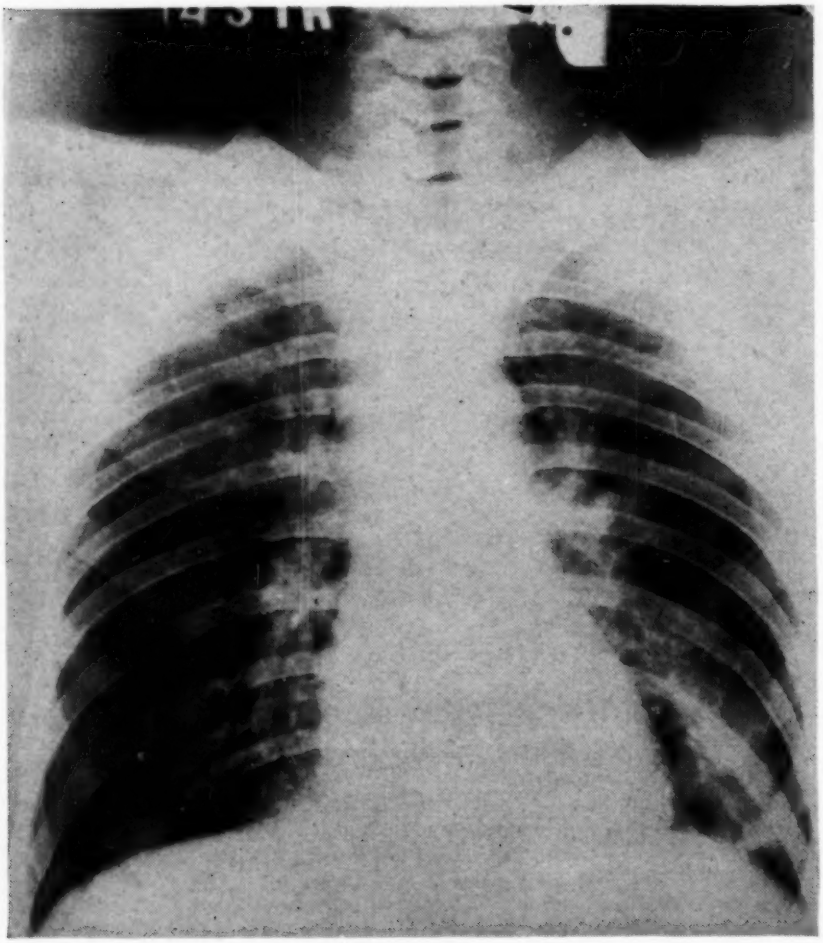


Fig. 15—*Broncho-alveolar Phase*, showing diffuse mottling in the left base and right cardiophrenic sinus; also patches of small discrete lesions in the right lower and right upper lobes and in the region of the seventh left interspace.

fluid was present, streptococci were present. In the other half the fluid was sterile.

At present it is our belief that the pleurisies and other complications such as nephritis, periarthritis, and intercurrent infections such as tonsillitis, sinusitis, myositis, thrombophlebitis and lymphadenopathy are due to secondary invaders.

SUMMARY AND CONCLUSIONS

In a study of some five hundred patients representing a cross section of an epidemic of acute respiratory tract infection numbering some six thousand cases, twenty-five per cent were found to have atypical pneumonia.

Atypical pneumonia should not be considered a disease entity but part of a syndrome in which the pulmonary lesions are but one manifestation of a generalized infection. So considered it might well be a physiological accident and not a pneumonia in the accepted sense of the term.

The pathology is described, evidence being advanced that it is an interstitial pneumonitis and that the pathology is similar to that found in other virus infections of the pulmonary tract.

The process is divided into four phases, namely, the bronchitic, the peribronchitic, the alveolar, and broncho-alveolar. A description of the physical and x-ray findings peculiar to each phase is given. Explanation for these findings is attempted by correlating them with the pathology.

RESUMEN Y CONCLUSIONES

En un estudio de unos quinientos pacientes representativos de una epidemia de infección aguda del aparato respiratorio que atacó a unos seis mil sujetos, se descubrió que el veinte y cinco por ciento tenían neumonía atípica.

No debe considerarse a la neumonía atípica una enfermedad de por sí, sino parte de un síndrome en el cual las lesiones pulmonares no son más que una manifestación de una infección general. Así considerada, es posible que no sea más que un accidente fisiológico y no una neumonía en el sentido que habitualmente se da a este término.

Se describe la patología y se aducen pruebas de que es una neumonitis intersticial y de que la patología es semejante a la que se encuentra en otras infecciones del aparato respiratorio causadas por virus.

El proceso se divide en cuatro fases, a saber: la bronquítica, la peribronquítica, la alveolar y la bronco-alveolar. Se da una descripción de los hallazgos físicos y roentgenográficos peculiares a cada fase. Se intenta la interpretación de estos hallazgos correlacionándolos con la patología.

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A New Era in the Fight Against Microbes*

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Ever since the role of microbes as a cause of disease in plants and animals was discovered, science has been engaged in a continuous and relentless warfare against pathogenic microorganisms. There were several principles employed successively, beginning with antisepsis and asepsis, later followed by serum- and vaccine-therapy, and finally by chemotherapy, which has held the ground ever since Wassermann's discovery of salvarsan. Gold and copper therapy belong in this class. A recent high of chemotherapy was reached with the development of the sulfonamides.

Chemotherapy has always had one serious drawback: Any chemical potent enough to inhibit the growth of bacteria or to kill them has a toxic influence upon the host. All chemotherapeutic remedies have toxic by-effects which necessitate their careful and judicious use. Their beneficial and toxic effects differ widely, according to individual differences in response and tolerance.

The most modern principle in fighting microbes is the utilization of substances created by the metabolism of soil-bacteria and fungi, which is now under intensive investigation by many scientists.

It all began in 1929 when Fleming,[1] in the University of London, found that a substance derived from a fungus (Penicillium notatum) had definite and potent bactericidal powers. This discovery was apparently forgotten until 1940, when Chain, Abraham, Florey,[2] and their associates in England, took the matter up again, and American scientists joined in the investigation.

In 1939, Dubos[3] of the Rockefeller Institute in New York discovered that the culture of a soil bacterium, when added to a viable culture of pyogenic cocci, killed these cocci, even if added in minute quantities. The phenomenon was produced by a substance excreted by two soil bacteria of the tyrothrix group, B. subtilis and B. brevis. This substance, called tyrothricin, consists of two components: one, the more powerful, called gramicidin, active against Gram-positive organisms only; the other, called tyrocidin, which also inhibits or kills Gram-negative bacteria. Gramicidin and tyrothricin have been investigated for two years,[4,5] and many experiments *in vitro* and *in vivo* have been carried out. They are most

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potent in direct contact with the offending organisms, as in ulcers, infected wounds, on the mucosa and in infected body cavities, like empyemas, the urinary bladder, the maxillary sinus, or the middle ear. Both substances are extremely hemolytic,[7] and small amounts injected intravenously into dogs and rabbits are fatal. Therefore, neither tyrothricin nor gramicidin can be used intravenously in man, although they are innocuous and highly effective on granulating surfaces.

A different story is told about penicillin, the fungus extract first described by Fleming. The reports by Herrell[8,9] on the use of penicillin in 10 cases, 8 of which were fulminant infections with staphylococcus aureus, show the remarkable action of this substance, which he used intravenously by the continuous drip method. The cures reported are little short of miraculous. This penicillin B, as made and purified by Roberts and associates at the University Hospital in St. Louis,[10] is non-toxic (except for mice) and non-hemolytic; therefore, can be used intravenously in septicemias, where it will render the bloodstream sterile in a few hours.

Florey and Florey, in a recent publication,[11] report fifteen cases of severe illness treated with penicillin orally, intramuscularly, intravenously and intrathecally. There were 10 severe staphylococcus infections, 1 sulfo-fast streptococcic meningitis, 3 cases of actinomycosis and streptothrix infection, all yielding very satisfactorily to the treatment. The only failure was a case of subacute bacterial endocarditis due to streptococcus viridans. One hundred seventy-two cases were treated locally—eye, mastoid, and chronic wound infections, and miscellaneous local septic conditions. In most cases the pyogenic infection was eliminated and healing followed. The authors emphasize that penicillin is bacteriostatic and not bactericidal, and that the body defenses have to destroy the pathogenic organisms, while the penicillin prevents multiplication of the infecting microbe. For general use the authors advocate the intramuscular route, injections to be given 3-hourly, 10,000-30,000 Oxford Units per dose. The continuous application is necessary, as the drug is rapidly excreted by the kidneys and a sufficient concentration must be kept up in the bloodstream in order to effect bacteriostasis.

The exceptionally prompt curative action of penicillin in 3 cases of sulfo-fast gonorrhea is reported by Herell, Cook and Thompson.[12]

Actually, the importance of these discoveries cannot be overemphasized. In trying to explain the discoveries and place the facts correctly into the scheme of things we have to go back to Louis Pasteur who investigated the medieval theory of "Miasma" as the cause of disease and pestilence. The scientists of the Middle Age called "Miasma" an unexplained substance or vapor exhaled by the earth in certain regions, detrimental to the health of humans and

animals living in such places. They assumed that the millions of corpses, buried at the time of the great epidemics which swept Europe so often, contaminated the soil with a great number of disease germs which were buried with them. Pasteur upon investigation found that disease germs, buried in the earth, did not survive long, but were exterminated by other microbes, harmless to man and beast, which inhabited the soil. Dr. Dubos, starting from these considerations, gradually developed a strain of soil bacteria which produced gramicidin, a substance highly active against many kinds of pathogenic microorganisms.

However, these discoveries of Fleming and Dubos suggest the existence of a major principle in nature, which we must try to understand in order to benefit by it fully, or at least to be able to map out the proper approach to the problem and the necessary investigations. It does not seem logical to assume that, of all the many types of soil bacteria, only one or two kinds should possess this remarkable antimicrobic power; and that of the thousands of soil molds known, only one strain should produce a substance of the remarkable potency of penicillin. Such limitation would not be in accord with the magnitude of the principle involved.

In following Pasteur's trend of thought, and enlarging upon it, we arrive at conclusions such as these:

The world of microorganisms seems grossly to be divided into two main branches. One of them, the habitants of the surface of this planet, is the bacteria and fungi of the soil, and the many one-cell organisms of the waters—plants, fungi, amoebae, etc. The other main branch of the microcosmos lives with or on the plants and animals which are on the surface of this globe, on the soil or in the water, the sea, the lakes and streams. Some of them are parasitic, symbiotic, and sometimes beneficial; many are pathogens. The members of these two microscopic worlds keep quite well to their realms; only a few instances are known where soil bacteria or fungi have transgressed their boundaries by becoming pathogenic, as for instance, Anthrax, Tetanus and Gas-bacilli, or Actinomyces, Streptothrix, etc. I do not believe that, vice versa, many of the pathogens have invaded the soil or the waters, although, especially in tropical and subtropical regions, the warm and humid soil of the jungles or marshes, the sun-baked shores of the sea, the rich and sheltered bottoms of the lakes and streams would provide excellent media for their growth. If we consider that for millions of years, every day, uncountable billions of pathogenic microbes have been incorporated in the soil and the water of this earth, as their diseased or dead plant and animal hosts returned to the soil and waters from whence they came—it is clear that if these pathogens had found a suitable habitat in the surface soils and waters and had adapted themselves

to conditions there, they would have lived and propagated—then millions of years ago the face of this globe would have become a huge, festering boil, a seething mass of poisonous microbes, miles thick, excluding any other form of life on this planet. However, that has not happened; and the only explanation is that there is a sufficient and powerful defense in the soil and waters to prevent any invasion of this realm by the pathogenic microorganisms. The metabolic products of soil bacteria and fungi are so powerfully bactericidal even in unbelievably high dilutions that a mass invasion of pathogens is impossible. Coming to know and to understand this, we realize that in our fight against microbes and their deleterious effect upon us, our animals and plants, we have the most powerful allies in the soil bacteria and fungi, allies which have been fighting the common enemy successfully for millions of years. Taking advantage of these forces, we shall have the most propitious chances the human race ever had, to succeed in the war on microbic disease.

In view of the foregoing considerations, it becomes evident that we are about to enter into a new era in our fight against microbes with this introduction of a completely new principle in medicine. It reminds me of the successful eradication of some imported plant pests, which finally was achieved only by importing their natural enemies from their original habitat.

The field is vast, and a tremendous amount of laboratory and clinical experimentation is being done and will have to continue for a long time in order to reap all the benefits that are in store for suffering humanity.

As I have suggested before, in order to develop still other such beneficial substances, most probably of different and more specific properties, we shall have to investigate many more soil-bacteria and many more fungi. Fisher,[13] reporting on the manufacture of a "crude" penicillin, states that he found at least one strain of penicillium, different from Fleming's original strain, which was equally or more active. Comparatively short investigations of my own have yielded one mycobacterium, other than B. subtilis or B. brevis, which produces a substance (M-Substance), in many respects similar to gramicidin. Several of the fungi which I have grown for years for use in diagnosis and treatment of fungus allergies, when cultured on a special medium, produce a substance highly bacteriostatic and also bactericidal *in vitro* and, upon local application, *in vivo*. Thus, I believe I have proven my point, and further search and experimentation undoubtedly will yield many more useful agents.

The widening of the scope of these sources of bactericidal substances is highly desirable in view of the scarcity of tyrothricin and penicillin, both of which are difficult to prepare. All the material

produced now is reserved for military use, and there is hardly any available for civilian use, either here or in England.

The importance of these discoveries for the treatment of chest diseases is obvious. Empyema, the dreaded complication of artificial pneumothorax and post pneumonic development, has been successfully sterilized with tyrothricin and M-Substance in a few days by several workers. Wound infection after thoracic surgery can be cleared up easily. There will be a way to use these anti-microbic substances in the treatment of bronchiectasis.

The experiments carried on so far showed that these substances are not active against the tubercle bacillus. However, the deductions from what little work has been done in this direction are not conclusive. I am convinced that the development of new and different fungus extracts will lead to important discoveries. Such substances, if found to be active against the Koch bacillus, will be found very useful in the modern treatment of tuberculous cavities.

SUMMARY

A new addition to chemotherapy is the modern principle of utilizing substances created by the metabolism of soil bacteria and fungi in fighting and destroying microbes. A review of the literature from 1929 is presented, and a suggestion made that other soil molds, in addition to penicillin, may possess anti-microbic characteristics. Several such fungi and soil bacteria are under cultivation now and there is hope that these extracts will lead to important discoveries.

RESUMEN

Una nueva adición a la quimioterapia es el principio moderno de utilizar sustancias producidas en el metabolismo de bacterias del suelo y de hongos, en combatir y destruir microbios. Se presenta un repaso de la literatura desde 1929 hasta el presente, y se indica que, además de penicilina, es posible que otros mohos del suelo posean características anti-micróbicas. Se están cultivando actualmente varios de estos hongos y bacterias del suelo, y se abriga la esperanza de que estos extractos nos traerán descubrimientos importantes.

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The Use of Sodium Hypochlorite in the Concentration of Tubercle Bacilli*

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Many different preparations containing sodium hypochlorite have been used as digestants of sputum for concentrating tubercle bacilli. Recently Oliver and Reusser[1] reported the use of Clorox which contains 5.25 per cent sodium hypochlorite as a digestant. They found this method simpler, more rapid and better than the direct smear, sodium hydroxide, chemical flocculation and papain methods. In addition Clorox is readily available and comparatively inexpensive.

Their method consists essentially of mixing equal amounts of Clorox and sputum and allowing to stand at room temperature for 10 minutes with occasional shaking. Specimens are centrifuged in 15 ml. conical tubes for 10 minutes at 3000 R. P. M. and then decanted and the tubes allowed to drain for 2 minutes. The sediment is transferred to a slide with an applicator, allowed to air dry and stained without heat fixing.

Because of the excellent results reported by Oliver and Reusser, we made a preliminary comparison of this method with the sodium hydroxide method in use on 35 sputa from tuberculous patients. The method used in mixing and splitting these specimens is described below. The examinations were made by two different workers, one making 1 minute counts and the other counting definite numbers of microscopic fields on each slide. Parallel results were obtained so only the 1 minute count will be considered. For specimens showing large numbers, the acid fast organisms were counted in multiples of 5 and recorded to the nearest 100 whereas if specimens showed less than 100 organisms, they were recorded to the nearest 5 and those having less than 5 organisms were recorded as the actual number found. Both methods were positive with 29 specimens. In 13 of these 29 specimens the number counted with the Oliver-Reusser method was from 1 to 5 times greater than the sodium hydroxide method, while 15 specimens showed about the same number of organisms and 1 was negative. Six specimens were positive with the Oliver-Reusser method and negative with sodium hydroxide.

In another series of 21 sputa, 17 were negative and 4 were positive

*The St. Louis Health Division Laboratories.

with both sodium hypochlorite and sodium hydroxide. The average field count for the 4 positive specimens was 1, 2, 4 and 5 for the sodium hydroxide and 10, 25, 40 and 45 respectively for the Oliver-Reusser method. Another 32 specimens were examined by direct smear and with Clorox. Both methods were negative with 25 specimens and positive with 7; however, the Oliver-Reusser method showed from 100 to 400 per cent more organisms.

This preliminary work on 88 sputa, 35 from tuberculous patients and 53 from unknown sources, showed the Oliver-Reusser method to be more sensitive. In order to secure additional data on the efficacy of Clorox, which contains 5.25 per cent sodium hypochlorite, in the digestion of positive sputum, a solution containing this amount of sodium hypochlorite was prepared in the laboratory. Another commercial product, Purex, containing 3 per cent sodium hypochlorite, was secured and a similar solution was also prepared.*

*These products were prepared by Miss Martha Herrmann of these laboratories.

TABLE I

COMPARISON OF CLOROX, PUREX, 5% AND 3% SODIUM HYPOCHLORITE IN THE CONCENTRATION OF SPUTUM FOR TUBERCLE BACILLI

Spec. No.	*Direct Smear*	*Clorox*	*Purex*	*5% NaOCl*	*3% NaOCl*
		Each Specimen Counted for 1 Minute			
1	200	200	300	200	300
2	30	100	100	50	50
3	100	400	400	100	100
4	200	400	200	70	80
5	40	200	200	60	100
6	70	100	100	100	200
7	90	300	300	100	200
8	5	10	15	5	10
9	10	5	35	30	60
10	3	10	30	5	35
11	5	5	10	10	15
12	10	55	55	60	55
13	5	100	90	35	100
14	2	15	15	80	50
15	10	200	200	150	100
16	2	55	40	30	25

These four products were compared using the Oliver-Reusser technique in examining aliquots of 16 positive specimens. Table I shows that all of these products are definitely more sensitive than the direct smear. Apparently Clorox, Purex and 3 per cent sodium hypochlorite are about equally effective. The 5 per cent sodium hypochlorite solution prepared in the laboratory was somewhat less effective. The average number of tubercle bacilli per slide counted for 1 minute for the direct smear and the four products (Clorox, Purex, 5 per cent and 3 per cent NaOC1) was respectively 49, 135, 131, 68 and 92.

Additional data on the comparative efficiency of Clorox and sodium hydroxide as digestants were secured on another series of specimens examined by the following four methods. Method A refers to the direct smear. Method B is the Oliver-Reusser technique. Method C consists of treating the sputum with 4 per cent sodium hydroxide, heating for 30 minutes at 37° C., diluting with salt solution and then centrifuging. Method D consists of adding a mixture of 2 per cent sodium hydroxide and 2½ per cent phenol to equal parts of sputum, autoclaving for 20 minutes at 15 pounds and then centrifuging.

Twenty-eight tuberculous sputa were agitated vigorously in a shaking machine* for 15 minutes and then direct smears were made from the homogenized specimens. Undoubtedly this homogenization increases the sensitivity of the direct smear. Three ml. of the remaining sputum was used for each of the 3 concentration methods. Table II shows that the Oliver-Reusser method is definitely the most sensitive of these. Most specimens showed twice as many tubercle bacilli with this method compared with the direct smear, while 2 specimens (Nos. 7 and 15) showed 40 times as many organisms. The average number of tubercle bacilli per slide counted for 1 minute for the A, B, C and D methods were respectively: 270, 600, 500 and 450.

Twelve specimens were prepared by adding small amounts of positive to large amount of negative sputum and shaking vigorously. Preparations were made by the four methods described above and the results are recorded in Table III. A 3-minute count was made on these specimens because of the small number of tubercle bacilli found. On practically all specimens, the Oliver-Reusser technique produced a larger number of acid fast organisms than did any of the other methods. The average number of tubercle bacilli counted for 3-minute periods with methods A, B, C and D were respectively: 14, 62, 14 and 25.

As a check on the technic of mixing and splitting of specimens

*International Equipment Company shaking machine No. B2428.

TABLE II

THE OLIVER-REUSSER SPUTUM CONCENTRATION METHOD COMPARED WITH THREE OTHER METHODS

	METHODS			
Spec. No.	*A*	*B*	*C*	*D*
		Each Specimen Counted for 1 Minute		
1	70	700	400	500
2	60	100	200	75
3	150	300	500	600
4	200	1000	1000	500
5	50	500	500	300
6	35	800	300	300
7	25	1000	300	400
8	600	600	600	600
9	200	300	200	40
10	200	500	500	500
11	600	1200	700	0*
12	300	800	600	500
13	300	700	900	400
14	500	900	500	500
15	25	1000	1000	600
16	100	300	300	300
17	100	400	300	200
18	100	700	300	100
19	300	500	700	800
20	50	400	200	400
21	400	800	500	500
22	300	500	400	300
23	400	500	100	500
24	400	1000	500	600
25	200	300	200	300
26	300	400	300	300
27	600	1000	700	1000
28	1000	1000	1000	1000

*Specimen washed off.
A=Direct smear.
B=Clorox method.
C=4% NaOH plus 30 minutes at 37° C.
D=2% NaOH plus Phenol and Autoclaving.

TABLE III

THE OLIVER-REUSSER CONCENTRATION METHOD COMPARED WITH THREE OTHER METHODS ON SPUTUM CONTAINING SMALL NUMBERS OF TUBERCLE BACILLI

	METHODS			
Spec. No.	*A*	*B*	*C*	*D*
		Each Specimen Counted for 3 Minutes		
1	4	50	10	5
2	3	20	5	4
3	10	10	5	5
4	70	100	60	100
5	2	10	1	3
6	3	15	—*	5
7	—*	3	2	2
8	2	3	1	1
9	35	300	40	150
10	10	65	10	35
11	30	150	25	50
12	4	15	4	1

*Tuberculosis not found after 10 minutes search.
A=Direct smear.
B=Clorox method.
C=4% NaOH plus 30 minutes at 37° C.
D=2% NaOH plus Phenol and Autoclaving.

for the comparative tests recorded in Tables II and III, 8 positive sputa were treated in the same manner as above and 3 aliquots of each specimen were prepared by the Oliver-Reusser method. Table IV shows that reproducible results are obtained and therefore the data secured in this study are statistically sound.

Oliver and Reusser stated that Clorox sterilizes tuberculous sputum because of their experiment in which 30 positive sputa after the digestion process gave no growth in culture on 42 days' incubation. In order to test this further, we inoculated guinea pigs after digesting 8 positive sputa (Nos. 21 to 28 in Table II) and found that none of the pigs developed tuberculosis after 10 weeks with the possible exception of the animal inoculated with specimen No. 26. This animal had gross pathology suggestive of tuberculosis although acid fast organisms were not demonstrated in any of the organs examined.

Many state laboratories place preservatives in empty sputum bottles that are distributed because the specimens that are returned in them may be delayed 2 to 3 days before reaching the laboratory.

From the limited experience described below it would seem that Clorox or a similar product could be used for this purpose. Ten positive sputa were treated with Clorox and then aliquots were removed after 10 minutes, 1, 2, 3 and 7 days. Table V shows that tubercle bacilli are preserved in Clorox for at least 1 week. One

TABLE IV

COMPARISON OF ALIQUOTS OF TUBERCULOUS SPUTUM USING THE OLIVER-REUSSER METHOD

	Aliquots of Same Specimens		
Spec. No.	*Each Specimen Counted for 1 Minute*		
	A	*B*	*C*
1	900	1000	1000
2	1000	1000	900
3	20	30	15
4	1000	1000	1000
5	50	75	60
6	150	200	200
7	5	5	5
8	50	75	50

TABLE V

THE EFFECT OF PROLONGED DIGESTION OF TUBERCULOUS SPUTUM WITH CLOROX

Spec. No.	*Digestion With Clorox*				
	10 Min.	*1 Day*	*2 Days*	*3 Days*	*7 Days*
1	65	200	100	200	200
2	300	500	500	Insufficient specimen	
3	25	25	Insufficient specimen		
4	2	—	Insufficient specimen		
5	400	300	400	500	400
6	100	50	50	Insufficient specimen	
7	200	200	300	400	
8	300	700	900	Insufficient specimen	
9	1000	1000	1000	1000	1000
10	200	500	800	Insufficient specimen	

advantage of this method is that the sputum would be thoroughly digested when it reached the laboratory. A disadvantage is that Clorox will dissolve cork or plastic liners used in some types of sputum bottles. However, such corks and plastic liners, as well as the wooden applicators used for smearing, when treated with Clorox for 24 hours, do not show acid fast organisms in the sediment.

CONCLUSIONS

The digestion of sputum with sodium hypochlorite was compared with direct smear and sodium hydroxide concentration methods. In the series of 144 sputa tested, the digestion by a commercial form of sodium hypochlorite was found to be more accurate and a more delicate method of concentrating sputum specimens. The commercial sodium hypochlorite appears to be of value as a preservative in sputum bottles for use by state laboratories.

CONCLUSIONES

Se comparó la digestión del esputo con el hipoclorito de sodio con las técnicas del frote directo, del hidrato de sodio, de la floculación química y de la papaína. En cada una de las cinco series comprobadas se descubrió que la digestión con una forma comercial del hipoclorito de sodio fue una técnica más exacta y más delicada de concentrar muestras de esputo. Parece que el hipoclorito de sodio comercial tiene mérito como preservativo en los frascos para esputo usados en los laboratorios de los estados.

REFERENCE

1 Oliver, J., and Reusser, T.: "Rapid Method for the Concentration of Tubercle Bacilli," *Am. Rev. Tuberc.*, XLV: 450, 1942.

Experiences in a Program for the Control of Pulmonary Tuberculosis in Chicago*

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The Municipal Tuberculosis Sanitarium has entire charge of the tuberculosis campaign in Chicago. The organization consists of a 1200-bed sanitarium, a 260-bed suburban branch, seven clinics and five field pneumothorax stations. As operative under the enabling legislation, the Glackin Act, all policies of both administration and treatment devolve on a board of three directors. The set-up, by placing sole responsibility in one board of directors, makes possible a unified plan of treatment and brings both sanitarium and field management into one general scheme. As will be seen later, this coordination of field and sanitarium activities is very effective in certain phases of the program.

GENERAL CONTROL MEASURES

The report of the case is, of course, compulsory. Follow-up and pre-sanitarium care are undertaken by the clinic staff of 34 physicians and 144 field nurses. On the whole, the patient cooperation is good. As elsewhere, however, the recalcitrant open case presents a problem. Nowhere, as far as the writer knows, has this problem of the non-cooperative open case been met adequately. Among many of the factors to hinder a solution may be mentioned tardy diagnoses, lack of hospital accommodations, the nature and duration of the disease and the reluctance of the lawmaking bodies to face tuberculosis squarely as a contagious disease.

In Chicago, operating under the State rules and regulations, no open case may be employed as a school teacher, school employee, nursemaid, food handler, barber, bartender, etc. As a matter of fact, however, and taking school teachers for one example, the regulations do not mean very much. Though we have tried repeatedly we have been unable, to date, to secure the compulsory examination of teachers; and this handicap, which also applies to other types of work nullifies the occupational paragraph.

On a factual basis and according to existent legislation, we are

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limited largely to the protection of children. According to the regulations, no child under sixteen years of age may live in the same home with a case of open tuberculosis. If the disobedient open case refuses to cooperate with the field nurse, the clinic physician calls in an attempt to effect a solution. If he fails, a placard is tacked up. If the placard fails and all other measures are futile, the patient is forcibly hospitalized to Cook County Hospital. The preliminary measures, however, are usually successful and forcible hospitalization is necessary in only very few instances. As of April 1st, there were in Chicago, 1,417 open cases in hospitals or sanatoria and 1,290 in their own homes. Of the 1,290 in their own homes, only 31 were shown as in contact with children and all these were in process of clearance at the time of the report.

It must be realized, however, that in a city like Chicago, even in the face of sound case-finding, a large fraction of open tuberculosis escapes supervision. It must be borne in mind, also, that a very considerable percentage of positive sputum cases come under observation at a late date after they already have had ample opportunity to do harm. Complete control of open tuberculosis in a city the size of Chicago would imply logically, three or four times the present number of beds, x-ray examination of all residents, rigid legislation and prompt isolation of every open case. In a democracy, however, we are not prepared to face such totalitarian control at this time.

COLLAPSE THERAPY

In recent years collapse therapy has become an instrument of control as well as a measure of treatment. As far back as 1931, Dr. Frederick Tice, President of our Board of Directors, in the Bulletin of that year emphasized what he called the Public Health Indication. Among other things, he said: "We can turn off the fountains of infection more thoroughly, more effectively by compressing a diseased lung than by isolating a patient."

In line with this trend of thought and faced with a serious bed shortage, Dr. Tice instituted a plan of field collapse designed to meet the problem of the patient who, for lack of accommodation, could not hope to have institutional care. Starting with a few dismissed cases from the sanitarium, in 1931, the extramural department today has 2,075 cases under supervision, 1,646 of whom are actually receiving pneumothorax.

As mentioned in opening, the authority delegated to the Board of Directors has made possible a coordinated and unified plan. Patients who are started on pneumothorax at the sanitarium are soon dismissed to the clinics; and pneumothorax patients in the field, who show complications or fail to make progress, are referred to the

sanitarium for phrenic interruption, pneumolysis, extrapleural pneumothorax, or thoracoplasty.

From the standpoint of control, the program may be considered satisfactory. In a study made in 1937, of 1,702 positive sputum cases treated with collapse and still living at the time of the report, 1,215, or 71.4 per cent were completely converted; 70, or 4.1 per cent, were incompletely converted; and 417, or 24.5 per cent, remained positive. These results are substantiated elsewhere and all signs seem to indicate that large scale collapse therapy will continue to be an effective measure of control. To be really effective, however, the large scale collapse plan must have certain essentials: sanatorium backing, a well-trained staff, close clinic supervision and nursing follow-up in the home.

CASE-FINDING

No control plan can exist on a poor case-finding foundation. Even at best, in metropolitan centers, a very serious percentage of tuberculosis will escape supervision. In case-finding we attempt to cut into this percentage as much as possible. In addition to the usual measure of case-finding, which cannot be discussed within this time limit, there are certain procedures in our experience which call for a word or two.

Tuberculin Testing in the Schools—Over a three-year period and throughout 1,087 schools, a total of 167,345 children were tested, of whom 27,401 proved positive. The work was confined largely to the kindergarten, first and eighth grades. In all, 98,000 elementary school children were tested. In the high schools 23,000 pupils were tested in freshman year, 19,000 in sophomore year, 12,000 in junior year and 11,000 in senior year. In the trade schools, junior colleges, and colleges 3,500 more students were tested. Roughly, 13 per cent of the elementary group, 22 per cent of the high school group, and 33 per cent of the college group showed a positive reaction.

Of the 167,000 children tested, 23,532 of the positive reactors were x-rayed and 218 cases of reinfection type tuberculosis were found of whom 109, or 50 per cent, were in the moderately advanced or far advanced stages. These figures, for the children x-rayed, represent an incidence of 0.36 per cent for the four to seven year group, an incidence of 0.84 for the males and 1.63 per cent for the females in the sixteen years and over period. Taking now the number of cases found against the total number of children tuberculin tested, the percentage of new cases found is discouragingly small. In the four to seven year group, one child in every 6,556 was diagnosed as reinfection type, in the age group eight to eleven, one in every 5,199, in the age group twelve to fifteen, one in every 660.

In addition to the 218 cases diagnosed on the first film, 107

additional cases were uncovered by later x-ray of children diagnosed "suspect" on the original film. Furthermore, 226 additional cases were found through follow-up of the positive reactors into the home. In all, then, 551 cases of pulmonary tuberculosis were found. In view of the time consumed, the volume of follow-up and the cost, the results were not considered satisfactory.

Analyzing the survey from the point of view of cost and including the high school and college groups, the expenditure for each case found was estimated at $511.00. In the kindergarten and first grade groups, the cost per case, including the follow-up, was estimated at $3,700.00.

One or two additional features of the survey are worth consideration. Crossing disease incidence against degree of reaction, 0.46 per cent of the one-plus children showed manifest tuberculosis, 1.07 per cent of the two-plus, 1.16 per cent of the three-plus, and 1.10 per cent of the four-plus children. Follow-up of the positives uncovered a peculiar situation. To promote impartiality and to keep the survey as a separate entity, the examinations were conducted without any reference to the clinic records. Later, in following the positive reactors into the homes, the apparent source of infection was found in 3,284 and in 3,058 instances the patients were already on the register. In over half the cases the established sources of infection were pulmonary deaths in patients registered with the clinics prior to death. The remainder were old pulmonary cases still living, some of them in institutions, many of them collapse cases turned negative, many others arrested cases at the date of last examination. Results merely confirmed the feeling of the Board that close supervision of tuberculosis contacts represented a much more practical effort than routine examination of school children.

As a result of this large volume experience and in view of the figures as quoted, we were led to the belief that the value of routine school testing can be very much over-emphasized. Since the cases found did not justify the expenditure in finding them. we discontinued this type of endeavor. Fortunately, about this time the miniature x-ray appeared over the horizon and we immediately swung over to this type of survey.

Work with the photo-roentgenographic unit started in 1940 and both the 4x5 and 35 mm. films were used. Our experience with the 35 mm. was not happy and practically the entire survey was conducted through the medium of the 4x5. Regarding procedure, the mobile unit was housed, for varying periods, in locations throughout the negro and slum areas. Later, relief clients were x-rayed and at the present time the unit is concerned chiefly with photo-roentgenographic examination of factory employees.

Up to May 1st, 112,000 applicants had been x-rayed, 74 per cent adults, 26 per cent children, 34 per cent white, 66 per cent negro. To date, 4,071 cases have been found, the rate for adults being 4 per cent, for children 0.3 per cent. On the basis of race, the whites examined showed roentgenological evidence of tuberculosis in 4.9 per cent of the cases as against 2.7 per cent of the negroes. Though this may appear rather strange, it must be realized that the work was conducted largely in slum areas with a very substandard white population. Needless to say, the percentage of tuberculosis found varies greatly with the type of individual examined. The factory survey, for instance, in which we are engaged at present, shows an incidence of less than one per cent.

Regarding stage of the disease, 52 per cent of the cases found were classed as minimal, 35 per cent as moderately advanced, 13 per cent as far advanced. On the whole and to date, we have been quite satisfied with the results of the photo-roentgenographic survey and according to all indications this type of work will remain as a permanent feature of the case-finding program.

BCG—The BCG program, as carried out by the Municipal Tuberculosis Sanitarium, is modeled on the work of Calmette and Guerin. This control effort has been operative in Chicago since 1934. The first two or three years were devoted entirely to research and animal experimentation. Around 1937, after careful research had demonstrated the harmlessness of the culture, vaccination of humans was begun. To date, over two thousand individuals in Chicago, mostly infants, have received the vaccine. An equal number of so-called controls or test cases have been kept under observation. Up to the present time, 5 of the control or non-vaccinated cases have developed tuberculous bronchopneumonia and 2 died. Several others in the non-vaccinated group showed x-ray evidence of tuberculosis, which may or may not prove serious as time goes on. Among the vaccinated group, on the other hand, not a single case to date has developed any sign of the disease.

SUMMARY

While there are many other features of management which cannot be discussed here, the main features of the control plan, as operative in Chicago, may be summarized as follows:

1) Close supervision of the open case with protection of contacts in every way possible, as soon as possible.

2) Pneumothorax in the field for every case which shows an indication.

3) Close liaison between the sanitarium and clinics so that field collapse and sanitarium collapse are mutually complementary.

4) Total x-ray examination of slum areas, relief agencies, fac-

tories, high schools and colleges through the medium of the photo-roentgenographic unit.

5) Continued efforts to secure legislation for compulsory examination of school teachers, food handlers—of all persons, in short, engaged in occupation which bring them in close contact with children, adolescents, or food produce.

With the exception of additional legislation and more rigid methods of enforcement, the program on the whole looks promising. Regarding new measures of legislative control, there are many *ifs, ands,* and *buts.* Apparently it is difficult to sell lawmakers the idea that tuberculosis is a contagious disease in the full sense of the word.

RESUMEN

Aunque existen muchas otras peculiaridades de administración que no es posible discutir en este informe, las principales características del plan anti-tuberculoso operativo en Chicago pueden resumirse en la forma siguiente:

1) Extricta vigilancia de los casos abiertos y protección de los contactos en toda forma posible, tan pronto como sea posible.

2) Neumotórax en dispensarios en todo caso en que exista la indicación.

3) Intima cooperación entre el Sanatorio y los dispensarios a fin de que el colapso en los dispensarios y el colapso en el Sanatorio sean recíprocamente complementarios.

4) Examen radiográfico de todo el mundo en los barrios bajos, agencias de socorro a los indigentes, fábricas, escuelas secundarias y universidades por medio de la foto-roentgenografía.

5) Esfuerzos contínuos para conseguir la aprobación de leyes que hagan obligatorio el examen de maestros de escuelas, traficantes en alimentos, de todas las personas, en una palabra, empleadas en ocupaciones que los ponga en contacto cercano con niños, adolescentes o productos alimenticios.

Con la excepción de leyes adicionales y métodos más rigurosos de observancia forzosa, el plan, en general, promete mucho. En lo que toca a nuevas medidas de control legislativo, existen muchos *"sis, ies* y *peros."* Aparentemente es difícil hacerles comprender a los legisladores que la tuberculosis es una enfermedad contagiosa en todo el sentido de la palabra.

Clinically Primary Tuberculosis of the Pericardium*

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Primary tuberculosis of the pericardium as a pathological entity is not uncommon. As an outstanding clinical picture, its occurrence is relatively infrequent. Tuberculous pericarditis is more often secondary to an infection of the neighboring structures, such as the lungs, pleura, mediastinal lymph nodes, or part of a generalized miliary tuberculosis. Osler found in 1,000 autopsies, 275 cases with tuberculous lesions, in 7 of which the pericardium was involved. Norris reported that of 7,219 autopsies in several Philadelphia hospitals, 1,780 showed tuberculous lesions, 82 of which had involvement of the pericardium. Harvey and Whitehill[1] recently reported 95 cases of tuberculous pericarditis that had been admitted to the Johns Hopkins Hospital in the past 45 years. General autopsy figures reveal that about 25 per cent of all patients had tuberculous lesions and of the latter about 0.8 to 1 per cent had tuberculosis of the pericardium. German statistics reveal an even higher incidence, 3 per cent of all tuberculous children showing pericardial involvement.

The occurrence of tuberculosis of the pericardium as a primary manifestation of tuberculosis is relatively rare. Riesman[2] first called attention to the "clinically primary" type of tuberculous involvement of the pericardium by which he meant that active lesions of the disease elsewhere in the body could not be demonstrated at the onset of symptoms. The occurrence of an anatomically primary tuberculous pericarditis, apart from involvement of the mediastinal or tracheobronchial glands, or other lesions elsewhere, has been questioned by some, and categorically denied by others. The advanced age of the patients presenting primary tuberculous pericarditis has been uniformly stressed in the literature.

Pericarditis as the first clinical manifestation of activity of a tuberculous process in childhood is almost unknown. It usually occurs as a part of a generalized miliary dissemination in which all organs are involved. A careful search of the literature has revealed the report of only one such case of clinically primary tuberculosis of the pericardium in children under 15 years of age. The case was that of a female child, aged 9 years, reported by Blatt and Green-

*From the Tuberculosis Service, and the Pathological Laboratories, Newark City Hospital, Newark, New Jersey. Necropsy findings reported by courtesy of Dr. Harrison S. Martland.

gard[3] whose initial symptoms was referable to a pericarditis. However, the autopsy findings showed a widely disseminated miliary tuberculosis.

The following case is of more than usual interest, not alone because of the infrequency of clinically primary tuberculous pericarditis and its great rarity in childhood, but for the classical clinical features of the disease that it presents.

CASE REPORT

History—F. B., a Negress aged 14, who entered Newark City Hospital on August 21, 1938, complained of shortness of breath, abdominal swelling, and edema of both lower extremities. The family history was not informative. She had had the usual childhood diseases.

The patient had been followed in the Chest Clinic for a period of five years. During that time, the Mantoux test was persistently positive, but repeated examinations and x-rays did not reveal any focus of tuberculosis.

She had been active and well up to six weeks before admission to the hospital, when she began to suffer from transient headaches and a persistent cough with little expectoration. There had been shortness of breath at that time, but it had become progressively worse until the patient was forced to her bed, where she was comfortable only in the upright position. About two weeks prior to admission to the hospital, she began to have profuse night sweats, noticing, too, that her abdomen and legs had become markedly swollen.

Examination—At the time of admission, the patient presented herself as a well-nourished young negress, in marked respiratory distress, with a temperature of 102° F., pulse rate of 140, respirations of 40 per minute and a blood pressure of 118/70 mm. of mercury. The chest gave a flat percussion note posteriorly from the angle of the scapula downward to the level of the lung bases on both sides. The heart was enlarged. The area of cardiac dullness was increased and continuous with that of pulmonic dullness so that there was a "girdle" of the flatness to percussion extending the entire circumference of the thorax at this level. The heart sounds were distant and muffled. No friction rub or murmur could be heard. The abdomen was distended. The liver was enlarged and very tender. There was evidence of some ascites. The thighs were painfully edematous.

The laboratory examination showed 3,400,000 red blood cells, 10,600 leukocytes to the c. mm., and a hemoglobin of 9.4 gm. The urine had a specific gravity of 1.029, a trace of albumin and no pus cells. The Wassermann examination was negative, as were the blood cultures. The sedimentation index of the blood varied from 61 to 110. There were no tubercle bacilli in the stained smear of the sputum from several daily specimens.

On the day after admission, about 500 cc. of clear yellow fluid was removed from the right pleural cavity. In the next few days, 150 cc. and 300 cc. of bloody fluid was removed from the pericardium. Following this the left pleural sac was tapped and 100 cc. of clear yellow fluid was obtained. By staining methods, no tubercle bacilli were found in the sediment of the fluids removed from the pleura or the pericardium. How-

ever, the fluid removed from the pericardial sac was injected into a guinea pig. A necropsy on this pig four weeks later showed typical miliary tuberculosis.

On admission, the roentgen ray picture showed a much enlarged cardiac shadow, the left border being visualized in the axilla, and fluid in both pleural cavities (Fig. 1). Neither in the pictures taken on admission nor in those subsequently taken was there any definite evidence of pulmonary tuberculosis. The electrocardiogram on several occasions showed signs of increased intrapericardial pressure with inverted T waves and low voltage Q R S in all standard leads.

Progress—Fever was present during her entire stay in the hospital. The highest temperature recorded was 104° F., and during the course, it varied between this and 99° F. with typical afternoon elevations. The patient was treated symptomatically with gradual subjective improvement, the fluid in the pleural and pericardial sacs reaccumulating slowly after the tappings. The dyspnoea and edema disappeared slowly until the patient was quite comfortable in bed, despite the fact that the cardiac shadow was still greatly enlarged. The patient was released involuntarily on Oct. 3, 1938, with the diagnosis of tuberculous pericarditis, six weeks after admission to the hospital.

On October 11, 1938, one week later, the patient was readmitted to the hospital with marked respiratory embarrassment. Physical examination and x-ray pictures again showed pleural and pericardial effusions with congestion of both lung fields. The temperature was 104° F., the pulse rate was 140 per minute and the respirations 30 per minute. Two pericardial taps were performed within a few days, and 40 cc. and 100 cc. of clear amber fluid were obtained. Smears for tubercle bacilli on the sediment of these specimens and concentrated sputa were negative. However,

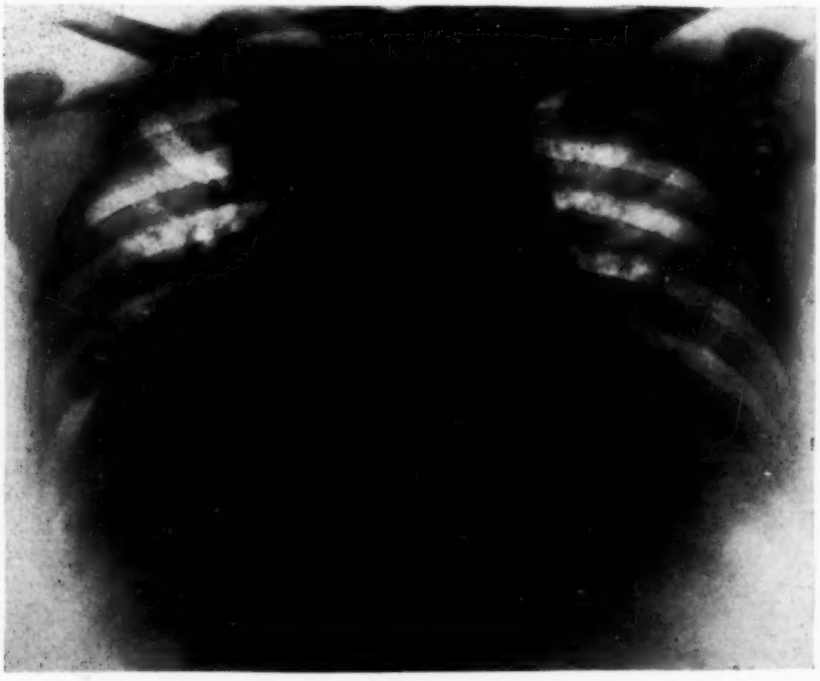

Fig. 1

the patient responded well to these tappings and complained only of continued headaches.

About two weeks after admission, signs of meningeal irritation appeared. The patient became semi-stuporous, muttering continuously, her eyes wandering aimlessly. A horizontal nystagmus appeared with rigidity of the neck; positive Kernig and Brudzinski signs could be obtained. A spinal tap was attempted and a blood-tinged fluid under slightly increased pressure was obtained.

Oral sulfanilamide therapy was begun and the patient received 15 gms. of the drug with sodium bicarbonate over a period of five days. On the fifth day, the patient appeared somewhat improved, acting brighter, her neck being less rigid. No Kernig or Brudzinski signs could be obtained. The only apparent residual signs were a bilateral persistent internal strabismus and some personality changes. The temperature curve had descended from 103° F. to 101° F. with little reduction in the pulse rate. However, despite intensive "prontylin" therapy, the patient again became stuporous after a few days, presenting increasing signs of meningeal irritation. She soon became incontinent, showed flaccid paralyses of both lower extremities with absent tendon reflexes, and died on November 9, 1938, about four months after the onset of symptoms. An autopsy was performed within twelve hours of the death under the direction of Dr. Harrison S. Martland.

A guinea pig innoculated with the spinal fluid removed on October 29, 1938, died six weeks later and autopsy revealed typical miliary tuberculosis.

Necropsy—Both layers of the pericardium were thickened to 3 mm., the parietal somewhat more than visceral. Between the layers were numerous small tuberculous nodules with typical miliary tubercles and giant cells. The heart was covered with a thick fibrinous exudate of "bread and butter" appearance and the sac was filled with about 200 cc. of clear amber colored fluid. There were no adhesions. There was no hypertrophy of the myocardium or dilatation of the chambers of the heart. The valves and myocardium were not involved.

There were 200 cc. of clear amber fluid in the right pleural sac and 100 cc. of a similar fluid in the left pleural cavity. The lungs were crepitant throughout. The mediastinum was thickened and several large, firm, discrete tuberculous lymph nodes were discovered. No evidence of a primary Ghon lesion in the lung parenchyma could be found.

The spleen, liver, adrenals, peritoneum, mesenteric lymph nodes and kidneys were normal.

The head was not examined.

COMMENT

The clinical picture of patients presenting primary tuberculosis of the pericardium is surprisingly uniform. The outstanding diagnostic features of the case reported were the insidious onset of the cardiac

failure, with fever and great increase of the cardiac outline, few symptoms referable to the chest, and the recovery of the tubercle bacilli in a bloody pericardial fluid. One striking note is the relative mildness of the complaints in contrast to the apparent seriousness of the physical findings. The failure to detect the enlargement of the tracheobronchial lymph nodes, which come to involve the mediastinal tissues and through these the pleura and pericardium, may be attributed to the marked enlargement of the cardiac outline. The absence of cardiac murmurs serves to distinguish this condition from rheumatic pericarditis with effusion.

Audibert and Olmer[4] stress the importance of making a diagnosis on the following cardinal signs: (1) Dyspnoea. (2) Increase in cardiac dullness. (3) Fever. (4) General appearance of weakness. The patients appear pale, tired and chronically ill. In addition, the heart sounds are distant and muffled; there is often a mild cough; there is moderate progressive edema; the liver is enlarged. They come to the conclusion that the diagnosis should be very seriously considered in: (1) A cardiac patient whose dyspnoea progresses relentlessly without ever receding. (2) A cardiac patient who has a persistent unexplained fever.

Death in this case may be attributed to a terminal tuberculous involvement of the meninges. In the younger cases the mode of exodus is by way of a terminal miliary dissemination, while in the older patients deaths are associated with inability of the cardiovascular system to cope with the added burden.

COMENTARIO

El cuadro clínico de pacientes que padecen de tuberculosis primaria del pericardio es asombrosamente uniforme. Las características salientes en el diagnóstico del caso presentado fueron: La iniciación insidiosa de la insuficiencia cardíaca, con fiebre y gran aumento en el tamaño del contorno del corazón, los pocos síntomas referibles al pecho y el hallazgo de bacilos tuberculosos en el líquido sanguinolento pericardíaco. Una peculiaridad notable es la relativa lenidad de los síntomas en contraste con la evidente gravedad de los hallazgos físicos. El hecho de que no se descubrió el crecimiento de los ganglios linfáticos traqueobronquiales, que llegaron a afectar los tejidos mediastinales y, a través de ellos, la pleura y el pericardio, puede atribuírsele al gran aumento en el tamaño del contorno del corazón. La ausencia de soplos cardíacos sirve para distinguir esta afección de la pericarditis reumática con efusión.

Audibert y Olmer[4] hacen hincapié sobre la importancia de hacer el diagnóstico basado en los signos cardinales siguientes: (1) Disnea. (2) Aumento en la submatidez cardíaca. (3) Fiebre. (4) Aspecto general de debilidad. Los pacientes presentan un aspecto pálido,

cansado y de sufrir de un mal crónico. Además, los ruidos cardíacos se oyen lejanos y apagados; a menudo existe una tos leve; hay edema moderado progresivo y hépatomegalia. Estos autores llegan a la conclusión de que debe considerarse seriamente este diagnóstico en: (1) El paciente cardíaco cuya disnea progresa implacablemente sin retroceder jamás. (2) El paciente cardíaco con una fiebre persistente inexplicable.

El desenlace fatal en este caso puede ser atribuído a la afección terminal tuberculosa de las meninges. Los pacientes más jóvenes fallecen debido a una granulia terminal diseminada, mientras que los pacientes de mayor edad mueren debido a que el sistema cardiovascular es incapaz de soportar la carga adicional.

REFERENCES

1 Harvey, A. M., and Whitehill, M. R.: "Tuberculous Pericarditis," *Medicine*, 16: 45-94 (Feb.) 1937.

2 Riesman, D.: "Primary Tuberculosis of the Pericardium," *Am. J. M. Sc.*, 122: 6 (July) 1901.

3 Blatt, M. L., and Greengard, J.: "Pericarditis as the Primary Clinical Manifestation of Tuberculosis in Childhood," *Am. J. Dis. Children*, 35: 631-636 (April) 1928.

4 Audibert, V., and Olmer, J.: "La Forme Subsigue Mortelle de la Pericardite Tuberculeuse Primitive Avec Epanchement de l' Adulte," *Presse Med.*, 38: 1121-1122 (Aug. 20) 1930.

The President's New Year Message

Twelve months ago, when the message for the New Year then beginning was being written, it was safe to say, with reference to the great assay by which our souls are being tried, no more than that the end seemed in the distant future, and that we knew not which of the scriptural times it would be for us—a time for tears or a time for laughter. That this was then the case, the nation's leader confirmed in his recent Christmas Eve message, when he said that this year, for the first time since the beginning of World War II, could the expression "Happy New Year" be more than wishful thinking. Let us hope, then, that this will be the last New Year greeting in which the reign of the Prince of Peace is spoken of as yet to begin.

Meantime, however, so long as the war continues to bulk large in our thoughts and daily work, there is some satisfaction in the rightful pride with which we consider the patriotic response of College members to the country's call. Those of us still in civilian life will do our best to carry on the so important work which now falls more heavily upon us; if, at times, the job seems a hard one, pride in our profession, and in our own peculiar contribution thereto will make us realize that "The labor we delight in physics pain."

The recent News Release from College headquarters showed a most gratifying response from the other American countries to the efforts of the College, working through its Council on Pan-American Affairs, to show that we do not lag behind the country at large in a desire to help professionally when possible, and to benefit by our progressive confreres' work in those countries. It is hoped that they have found useful the Spanish summaries of important articles in *Diseases of the Chest*.

It is not meant as a paradox or an attempt at turning a witty phrase to say that the physician works best in peace, yet is ever, through his own desire, at war. War, as the world sees it, is the horrible, yet limited-in-time period through which we are now passing, by God's help, victorious. But the physician's war against disease, which he prosecutes most effectively only when the other kind of war ends, goes on ceaselessly. Our Council on Military Affairs and Public Health is fighting, on the home front, the battle against tuberculosis in the industrial army, and this battle will go on long after the guns of World War II have been stilled. World planning is the order of the day, and world planning for tuberculosis control our own special contribution thereto.

I should like to give here brief appreciative mention to the Editor of *Diseases of the Chest* and his associates, on the completion of the magazine's first year in its new format, complimentary letters about which have been received from scores of subscribers; and also to thank the members of the College for their faithful attendance at Chapters meetings, a source of unusual satisfaction in these days of difficult travel and increased professional burdens.

We are ceaselessly reminded, as the war appears to draw toward its close, of the fearful responsibilities developing upon the statesmen who make the peace—and of the possibilities for fatal defects and blunders, as well as for vast beneficient progress. The physician's responsibility toward the post-war world is no less important, but his capability to achieve as yet unheard of progress is far greater and infinitely more assured than the statesman's, if only he will be true to himself and his profession. That each member of the College, devoting himself heart and soul to the tenets of the *Religio Medici*, will do his share to achieve this progress, and enjoy the peace of mind and heart which comes from a job well done—the great Osler's *aequinimitas*—is the New Year's wish of your President.

Dr. J. Winthrop Peabody, F.C.C.P.

COLLEGE NEWS

FALL MEETING OF THE BOARD OF REGENTS

The fall meeting of the Board of Regents of the American College of Chest Physicians was held at the Hotel Gibson, Cincinnati, Ohio, on November 17, 1943. The meeting of the Board of Regents was preceded by a meeting of the Executive Council of the College.

1944 Annual Meeting

The Board of Regents of the College voted to hold the tenth annual meeting of the College at Chicago, Illinois, June 10-12, 1944. The meeting, as provided in the By-laws of the College, will tie in with the annual meeting of the American Medical Association, which will meet in Chicago, June 12-16, 1944. The College headquarters will be at the Hotel Stevens in Chicago.

Fellowship Examinations

The Board of Regents ruled that applicants for Fellowship in the College approved by the Governors and Regents of the College and who have also been approved by a recognized medical specialty board, be admitted to Fellowship in the College without further examination. A request to this effect should be made to the Chairman of the Board of Regents in writing by the Regent of the College in the district wherein the applicant resides.

Appointment of New Governor

Dr. Hubert A. Boyle, F.C.C.P., Middleton, Massachusetts, was appointed by the Board of Regents to complete the unexpired term of Governor of the College for the state of Massachusetts left vacant because of the death of Dr. Frank H. Washburn.

Specialty Board for Diseases of the Chest

Following a lengthy discussion, the committee established by the Board of Regents to study the advisability of establishing a board for diseases of the chest was authorized to formulate a program relative to the establishing of a board on diseases of the chest, and that the plans for such a program be submitted by the committee to the Advisory Board of Medical Specialties. A further report on the activities of this committee is to be submitted to the Board of Regents at the meeting of the College in June, 1944.

Budget Revision

It was voted to include the sum of $100.00 in the present budget of the College for the purpose of purchasing subscriptions to special medical journals for the College library.

War Bonds

The Treasurer of the College was authorized by the Board of Regents to invest $10,000.00 of College funds in United States War Bonds.

Life Membership

The Secretary-Treasurer of the College was appointed by the President to prepare a plan for extending life membership to College members. This plan when completed will be submitted to the Board of Regents of the College for approval at the annual meeting in June, 1944.

College Medal

It was proposed that a fund be established for a medal to be presented annually for outstanding achievement in the specialty of diseases of the chest. The President was authorized to direct the Historian of the College to make suitable arrangements for the establishing of this fund.

Postgraduate Courses

It was proposed that a postgraduate course on diseases of the chest be made a part of the annual meetings of the College. The Illinois Chapter of the College was extended an invitation to prepare the first postgraduate course on diseases of the chest for presentation in connection with the tenth annual meeting of the College to be held in Chicago, June 10-12, 1944.

X-Ray Films of Army Rejectees

It was proposed that x-ray films of rejectees for military service be filed with the state departments of health, and that an interpretation of the films be sent to the local health officers. It was further proposed that the College take an active interest in this activity.

The following members of the Board of Regents attended the meeting:

Dr. J. Winthrop Peabody, F.C.C.P., Washington, D. C.,
Dr. George G. Ornstein, F.C.C.P., New York City, New York,
Dr. Jay A. Myers, F.C.C.P., Minneapolis, Minnesota,
Dr. Joseph C. Placak, F.C.C.P., Cleveland, Ohio,
Dr. Paul H. Holinger, F.C.C.P., Chicago, Illinois
Dr. E. W. Hayes, F.C.C.P., Monrovia, California,
Dr. Charles M. Hendricks, F.C.C.P., El Paso, Texas,
Dr. George B. Gilbert, F.C.C.P., Colorado Springs, Colorado,
Dr. Louis Mark, F.C.C.P., Columbus, Ohio,
Dr. Karl Shaffle, F.C.C.P., Asheville, North Carolina,
Dr. H. I. Spector, F.C.C.P., St. Louis, Missouri
Dr. James H. Stygall, F.C.C.P., Indianapolis, Indiana.

The meeting followed a dinner for the Governors and Regents of the College. The Cincinnati members of the College were hosts at a cocktail party preceding the dinner.

Dr. J. Winthrop Peabody, F.C.C.P., Washington, D. C., President of the College, presided at the meeting.

SOUTHERN CHAPTER ORGANIZED

On November 18, 1943, meeting conjointly with the Southern Medical Association at Cincinnati, Ohio, the Southern Chapter of the American College of Chest Physicians was officially organized. The following officers were elected:

President, Dr. Paul H. Ringer, F.C.C.P., Asheville, N. C.
First Vice-President, Dr. Alvis E. Greer, F.C.C.P., Houston, Texas
Second Vice-President, Dr. Carl C. Aven, F.C.C.P., Atlanta, Georgia
Secretary-Treasurer, Dr. Benjamin L. Brock, F.C.C.P., Waverly Hills, Kentucky.

The Constitution and By-Laws for official chapters of the College were adopted and the annual dues for membership in the Southern Chapter of the College were voted to be $1.00. All members in good standing in the national organization residing in the District of Columbia and in the following sixteen states are eligible for membership in the Southern Chapter of the College:

Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, Missouri, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, and West Virginia.

Short talks were made by Dr. J. Winthrop Peabody, F.C.C.P., Washington, D. C., President of the College, and Dr. Evarts A. Graham, F.C.C.P., St. Louis, Missouri. Dr. Benjamin L. Brock, F.C.C.P., presided at the meeting.

The Southern Chapter is the sixteenth chapter of the American College of Chest Physicians. The Chapter will meet annually with the Southern Medical Association and present scientific programs on diseases of the chest.

CONFERENCE OF CHAPTER OFFICIALS

The first Conference of College Chapter officials was held at the Gibson Hotel, Cincinnati, Ohio, November 18, 1943, and the following chapter officials participated in the Conference:

	Chapter
Dr. J. Winthrop Peabody, F.C.C.P., Washington, D. C., President, American College of Chest Physicians	
Dr. Minas Joannides, F.C.C.P., Chicago, Illinois	Illinois
Dr. M. H. Draper, F.C.C.P., Fort Wayne, Indiana	Indiana
Dr. James L. Mudd, F.C.C.P., St. Louis, Missouri	Missouri
Dr. Clyde M. Fish, F.C.C.P., Pleasantville, New Jersey	New Jersey
Dr. Irving Willner, F.C.C.P., Newark, New Jersey	New Jersey
Dr. James H. Donnelly, F.C.C.P., Buffalo, New York	New York State
Dr. Nelson W. Strohm, F.C.C.P., Buffalo, New York	New York State
Dr. Ross K. Childerhose, F.C.C.P., Harrisburg, Pennsylvania	Pennsylvania
Dr. Carl C. Aven, F.C.C.P., Atlanta, Georgia	Southern
Dr. M. D. Bonner, F.C.C.P., Jamestown, North Carolina	Southern
Dr. Benjamin L. Brock, F.C.C.P., Waverly Hills, Kentucky	Southern
Dr. Leo F. Hall, F.C.C.P., State Park, South Carolina	Southern
Dr. Paul H. Ringer, F.C.C.P., Asheville, North Carolina	Southern
Dr. Paul A. Turner, F.C.C.P., Louisville, Kentucky	Southern
Dr. Alvis E. Greer, F.C.C.P., Houston, Texas	Texas

Dr. Minas Joannides, F.C.C.P., Chairman of the Conference, presided, and Dr. M. D. Bonner, F.C.C.P., acted as secretary of the Conference. Following the reading by the chairman of a prepared report (to be published), a number of proposals were introduced by chapter officials and

discussed. Many of the proposals were recommended favorably to the Board of Regents of the College for adoption. These proposals are being prepared for the members of the Board of Regents of the College and they will be voted upon at the next meeting of the Board to be held at Chicago, June 10-12, 1944.

It was voted to organize a Conference of Chapter Officials to meet at the annual meetings of the College. The next Conference is scheduled to be held at Chicago, June 10-12, 1944. All of the College Chapters are expected to be represented at this conference.

PROGRAM ON DISEASES OF THE CHEST

SOUTHERN MEDICAL ASSOCIATION

Under the direction of the Scientific Program Committee of the American College of Chest Physicians, nine papers on various subjects related to diseases of the chest were presented at the thirty-seventh annual meeting of the Southern Medical Association held at Cincinnati, Ohio, November 17-18, 1943. The following speakers and discussants participated in the program:

Colonel Arden Freer (MC), F.C.C.P., Washington, D. C.,
Lieutenant Colonel Carl Tempel (MC), F.C.C.P., Washington, D. C.,
Lieutenant Commander D. F. Smiley (MC-V (s) USNR), Washington, D. C.,
Dr. Evarts A. Graham, F.C.C.P., St. Louis, Missouri,
Dr. Chester A. Stewart, New Orleans, Louisiana,
Dr. Paul H. Holinger, F.C.C.P., Chicago, Illinois
Dr. Arnold S. Anderson, F.C.C.P., St. Petersburg, Florida,
Dr. J. D. Riley, F.C.C.P., State Sanatorium, Arkansas,
Dr. Richard Davison, F.C.C.P., Chicago, Illinois
Dr. William Reinhoff, Baltimore, Maryland,
Dr. George G. Ornstein, F.C.C.P., New York, New York
Dr. James L. Mudd, F.C.C.P., St. Louis, Missouri
Dr. Maurice G. Buckles, F.C.C.P., Louisville, Kentucky,
Dr. Dean B. Cole, F.C.C.P., Richmond, Virginia,
Dr. Charles P. Cake, F.C.C.P., Washington, D. C.,
Dr. James L. Bibb, F.C.C.P., Chattanooga, Tennessee,
Dr. Hillis L. Seay, Huntersville, North Carolina,
Dr. Herman E. Hilleboe, F.C.C.P., Washington, D. C.,
Dr. Paul H. Ringer, F.C.C.P., Asheville, North Carolina
Dr. Roy A. Wolford, F. C. C. P., Washington, D. C.,
Dr. H. I. Spector, F.C.C.P., St. Louis, Missouri,
Dr. Alvis E. Greer, F.C.C.P., Houston, Texas,
Dr. Sydney Jacobs, F.C.C.P., New Orleans, Louisiana,
Dr. David T. Hyatt, F.C.C.P., Little Rock, Arkansas.

Dr. M. Jay Flipse, F.C.C.P., Miami, Florida, and Dr. Carl C. Aven, F.C.C.P., Atlanta, Georgia, presided at the Scientific Sessions.

Scientific Program Committee

Dr. Jay A. Myers, F.C.C.P., Minneapolis, Minnesota, Chairman,
Dr. C. Howard Marcy, F.C.C.P., Pittsburgh, Pennsylvania,
Dr. William A. Hudson, F.C.C.P., Detroit, Michigan.

MEXICAN CHAPTER

Reading from left to right: First row—Drs. Hector Martinez de Alba, Miguel Jimenez, Donato G. Alarcon, Edgar Mayer, Ismael Cosio Villegas, Octavio Bandala. Second row—Drs. Ricardo Tapia Acuna, Fernando Rebora, Aradio Lozano, Jesus Benitez, Fernando Katz, Jose Reynal and Horacio Rubio Palacios.

The Mexican Chapter of the American College of Chest Physicians was organized in Mexico City on September 9, 1943, with twenty-three charter members. In the above photograph are shown the members of the College in Mexico City attending the meeting. The following officers were elected:

President, Dr. Donato G. Alarcon, F.C.C.P., Mexico City,
Vice-President, Dr. Ismael Cosio Villegas, F.C.C.P., Mexico City,
Secretary-Treasurer, Dr. Octavio Bandala, F.C.C.P., Mexico City.

Dr. Edgar Mayer, F.C.C.P., New York City, New York, represented the American College of Chest Physicians at the meeting.

College Officials Visit Mexico

Dr. Chevalier L. Jackson, F.C.C.P., Philadelphia, Pennsylvania, Chairman of the Council on Pan-American Affairs of the American College of Chest Physicians, and Dr. Paul H. Holinger, F.C.C.P., Chicago, Illinois, Secretary-Treasurer of the College, visited Mexico City during December. They were guest speakers at a joint meeting of the Mexican Chapter of the College and the Society for the Study of Tuberculosis.

Eighth Postgraduate Course

During the months of September and October the Eighth Postgraduate Course on Tuberculosis was held at the Tuberculosis Sanatorium of Public Welfare at Huipulco, Mexico, D. F. Dr. Leo Eloesser and Dr. William B. Neff of Stanford University were invited as guest professors by the government in Mexico to give the lectures and demonstrations.

BRAZILIAN CHAPTER

The Brazilian Chapter of the American College of Chest Physicians, recently organized, announce the election of the following officers:

President, Dr. Samuel Libanio, Rio de Janeiro
Vice-President, Dr. Galdino Travassos, Rio de Janeiro
Executive Secretary, Dr. Carvalho Ferreira, Rio de Janeiro
Secretary-Treasurer, Dr. Affonso Mac-Dowell Filho, Rio de Janeiro

Dr. Samuel Libanio, President of the Brazilian Chapter of the College, is Director of the "Servicio Nacional de Tuberculose." Professor Affonso Mac-Dowell, Regent of the College for Brazil, presided at the meeting.

Dr. Affonso Mac-Dowell Filho, Secretary-Treasurer of the Brazilian Chapter of the College, reports that Brazil is planning on sending a delegation to the annual meeting of the College to be held in Chicago, in June, 1944. The first monthly meeting of the Brazilian Chapter was held in Rio de Janeiro, December 22, 1943.

CUBAN CHAPTER

The Cuban Chapter of the American College of Chest Physicians held a meeting on November 9, 1943, at the Instituto de Vias Respiratorias, Havana, Cuba. The following program was presented:

1) Report on the 48th annual Meeting of the American Academy of Ophthalmology and Oto-Rhino-Laryngology held at Chicago, Illinois, Dr. Pedro Hernandez Gonzalo, Delegate of the Chapter.

2) Cystic Disease of the Lung, Dr. Teodosio Valledor.

3) Clinical Reports:
Cicatricial fibro-stenosis of trachea, Dr. Rufino Moreno
Basal Atelectasis, Dr. Antonio Navarrete, F.C.C.P.

These papers were discussed by Drs. A. Codinach, J. Garcia Arrazuria, F. J. Menendez and Casana. Dr. A. Codinach was recently elected as an Associate Member of the American College of Chest Physicians.

OHIO CHAPTER

The Ohio Chapter of the American College of Chest Physicians conducted a luncheon meeting at the Hotel Gibson, Cincinnati, Ohio, November 17, 1943. The meeting was held in conjunction with the thirty-seventh annual meeting of the Southern Medical Association. A paper entitled, "A Study of Rejectees for Thoracic Abnormalities," was presented by Dr. William A. Hudson, F.C.C.P., and Dr. David Brachman, F.C.C.P., Detroit, Michigan. The following officers were elected:

Myron D. Miller, M.D., Columbus—President,
Raymond C. McKay, M.D., Cleveland—Vice-President,
Elmer E. Kirkwood, M.D., Youngstown—Secretary-Treasurer.

Dr. D. W. Heusinkveld, F.C.C.P., Cincinnati, Ohio, retiring President of the Ohio Chapter of the College, presided at the meeting.

PUERTO RICO CHAPTER

The annual meeting of the Puerto Rico Chapter of the American College of Chest Physicians was held at San Juan, P. R., December 12, 1943. The meeting convened at the School of Tropical Medicine under the chairmanship of Dr. David Garcia, F.C.C.P. The following papers were read at the scientific session:

"Primary Atypical Pneumonia," Dr. M. Guzman Rodriguez, F.C.C.P.
"Rest in the Treatment of Tuberculosis," Dr. A. Acosta Velarde, F.C.C.P.
"Carcinoma of the Lung," Dr. David Garcia, F.C.C.P.
"Tuberculous Meningitis," Dra. Alice Reinhardt, F.C.C.P.

The administrative session of the Chapter was then held and the report of the Secretary-Treasurer was approved. The Nominating Committee proposed that all of the members of the Board of Directors be re-elected. This motion was unanimously approved. During the next year the officers and Board of Directors will be as follows:

President, Dr. David Garcia, F.C.C.P.
Vice-President, Dr. J. A. Franco Soto
Secretary-Treasurer, Dr. A. M. Marchand, F.C.C.P.

Board of Directors

Dr. M. Godreau, F.C.C.P.; Dr. M. Santiago, F.C.C.P.; Dr. M. Guzman Rodriguez, F.C.C.P.; Dr. A. Acosta Velarde, F.C.C.P.

Following the Chapter meeting the members adjourned to the amphitheatre of the Hospital of the School of Tropical Medicine to hear a conference on pneumonia by Dr. Hobart A. Reimann, Jefferson School of Medicine, Philadelphia. A luncheon was served at the building of the Department of Health after the conference.

NEW JERSEY CHAPTER

The New Jersey Chapter of the College held its Winter Meeting at the City Hospital, Newark, New Jersey, December 8, 1943. The meeting was attended by 41 members of the College and guest physicians, in addition to hospital internes and the nurses from the Tuberculosis Division of the Newark Board of Health.

Harry Walburg, LL.B., one of the senior members of the firm of Cox & Walburg, read a paper on "Medico-Legal Aspects of Pulmonary Disease." His paper and his resume on the effect of trauma, dust and chemicals on the lungs, and the interpretation of each in accordance with the laws of the State of New Jersey was interesting and instructive.

Harrison M. Martland, M.D., Professor of Forensic Medicine, New York University, spoke on "Lung Pathology." He presented many pathological specimens and colored slides to illustrate his lecture, and he discussed a number of unusual cases from autopsies which he performed as the County Physician of Essex County and Pathologist at the Newark City Hospital. He then presented an unusual case from the Tuberculosis Service of the City Hospital and this case was fluoroscoped before the assembly.

In the absence of Dr. Clyde M. Fish, F.C.C.P., Pleasantville, President of the New Jersey Chapter of the College, Dr. Irving Willner, F.C.C.P., Newark, Secretary-Treasurer of the Chapter, presided at the Scientific Assembly and introduced the speakers.

REPORT OF THE SECRETARY-TREASURER*

At the last annual meeting of the College held at Atlantic City, June, 1942, the Auditor's Report for the fiscal year ending April 30, 1942, was presented and that report showed the total assets of the College as $16,445.45. Of this amount, $15,560.00 was in current assets and $885.34 for furniture and fixtures. Funds in the amount of $4,006.00 for Fellowship fees were maintained in a Special account. The net worth of the College on April 30, 1942, was $12,439.35.

At the last meeting of the Board of Regents held in Chicago, February 14, 1943, it was voted that the annual meeting of the College for 1943 be cancelled. The annual report of the Secretary-Treasurer of the College which was to be read at that meeting is accordingly presented herewith:

Auditor's Report for the fiscal year ending April 30, 1943.

ASSETS

Current Assets:		
Cash in Bank	$20,662.11	
Cash on Hand	25.00	
Receivables:		
For Reprints, Advertising and Subscriptions	356.50	
TOTAL CURRENT ASSETS		$21,043.61
Fixed Assets:		
Furniture and Fixtures		979.13
TOTAL ASSETS		$22,022.74

LIABILITIES AND NET WORTH

Liabilities:		
Collections toward Fellowships	$ 2,790.00	
Fellowship Fees Collected (Pend. Exam.)	350.00	
Special Fund	905.00	
Chapter Funds	5.00	
Reserve for Victory Tax	25.54	
TOTAL LIABILITIES		$ 4,075.54
Net Worth:		
Balance April 30, 1942	$12,439.45	
Net Income for Year	5,507.75	
		17,947.20
TOTAL LIABILITIES AND NET WORTH		$22,022.74

*Presented before the Meeting of the Board of Governors and Regents, Cincinnati, Ohio, November 17, 1943.

I am pleased to report that the College showed an increase in its assets for the fiscal year ending April 30, 1943, of $5,507.75.

On October 31, 1943, the College had a bank balance of $21,662.59. Against this are listed the following special funds:

Advance Payments for Fellowship Fees	$3,380.00	
Fees from Fellows Pending Examination	1,750.00	
Fees Accompanying Pending Applications	1,095.00	
TOTAL		$6,225.00

Most of these funds will become net assets as the applications are approved.

The College is operating during the current fiscal year on a budget of $20,968.33. This budget was submitted to the members of the Executive Council of the College on April 15, 1943, and approved by them. For the first six months of the present fiscal year, the statement as of October 31, 1943, shows that $9,279.10 of the Budget has been expended leaving a balance for the second six month period of $11,689.23.

Diseases of the Chest

Receipts from advertising and subscriptions for our journal, *Diseases of the Chest*, for the year ending December 1943 total $5,320.73. The cost of printing, paper stock, engravings, and the mailing of the journal was $4,579.58, a balance of $741.15 in excess of expenditures.

Dr. Paul H. Holinger, F.C.C.P.
Secretary-Treasurer.

REPORT OF THE MEMBERSHIP COMMITTEE*

At the beginning the Committee was impressed with the fact that the purposes of the American College of Chest Physicians should be made known to as many as possible of those physicians in the United States who were devoting all or the greater portion of their time to diseases of the chest. Also, it was necessary for the College, if it were to fulfill its purposes, to gain the active interest and support of all qualified chest physicians in the United States in developing a cohesive, cooperative, and articulate group, the aim of which would be, through their association and the certain amount of competition that evolves from such association, to improve the qualifications of the individual members and of the College as a whole.

It is, of course, the prerogative and responsibility of the Board of Regents to decide as to which specialists may be considered for admission to the American College of Chest Physicians, and in this connection the Membership Committee was informed that fellowship or associate membership in the American College of Chest Physicians was available to qualified physicians in high standing who devote all of their time, or a major portion of their time, to the treatment, teaching, or research in diseases of the chest; same to include thoracic surgeons, radiologists with

*Presented before the meeting of the Board of Governors and Regents, Cincinnati, Ohio, November 17, 1943.

proper training in chest diseases, bronchoscopists, otolaryngologists and pathologists who have had special training in tuberculosis.

Consideration had been given also to the inclusion of allergists and of heart specialists in this group, but no definite decision as to either group was arrived at so far as is known.

It was apparent that if the College were to become firmly established in the esteem of the medical profession of the United States, high standards for candidates were an essential requirement and should be adhered to. The accomplishments of the College would depend obviously upon the character of its membership and it must compare favorably in all particulars with similar professional groups.

The standards for fellowships and associate memberships having been decided upon by the Board of Regents, it was then the responsibility of the Membership Committee to contact as many as possible of the physicians in the United States who might be qualified for either fellowship or associate membership. It would be the responsibility of the Governor for each area to recommend as to the suitability of the candidate, this being a matter concerning which the Membership Committee would have no authoritative knowledge, except possibly in individual cases.

Accordingly, the Membership Committee after some correspondence among themselves as to the best method of approaching the problem, decided that the Membership Committee of the American College of Chest Physicians was appointed by the President of the College with its prime duty to increase the membership; that it was not its function to pass upon individual applicants; the machinery within the College for the passing upon applications and admitting qualified members in the College was well organized and had been functioning satisfactorily for a number of years.

All invitations extended to prospective members by our Committee were naturally to be made with the proviso that the applicant would meet the minimum requirements of the College for such membership. If and when an application was received at the executive office of the College, notices were mailed by that office to all Fellows of the College in the locality wherein the applicant resided. After a period of two weeks had elapsed, any comments received with regard to the applicant were attached to the papers and the same mailed to the Governor of the College in the state where the applicant resided. If the Governor passed favorably upon the applicant, the application was then mailed to the Regent of the College in the district where the applicant resided. Should the Regent act favorably, it was then mailed to the Chairman of the Board of Regents of the College for his approval. In addition to this, all applicants for fellowship were required to pass an entrance examination unless the same was waived by the Board of Regents of the College. This procedure, it was believed, provided a sufficient safeguard against admitting candidates who did not meet the minimum requirements for membership in the College. It was, therefore, the task of the Membership Committee to interest prospective candidates in the work of the American College of Chest Physicians and to urge such prospective candidates to make application for membership in the College.

1. Lists of prospective candidates were obtained from various sources:
   (a) Medical specialty directories.
   (b) Questionnaires with regard to medical specialists.
   (c) Various Government agencies.

(d) Medical societies.

(e) Lists submitted by Governors and Regents of the College.

All of this data was compiled by the executive office of the College in Chicago.

2. Copies of our journal, "Diseases of the Chest," were then mailed to each of the prospective candidates in order that they might obtain an insight into some of the College activities.

3. This was followed by a letter of invitation to make application for membership mailed on the official Membership Committee stationery, and signed by the Chairman of the Committee.

For instance, the members of the American Broncho-esophagological Association were circularized, some 112 invitations were mailed. A very satisfactory number of applications were received as a result. At one time we sent invitations to 200 chest specialists who were members of the military service and not at that time members of the College, each of these having previously been furnished a copy of our journal.

The Committee obtained all available names from the various agencies mentioned above, and these have been followed through in the manner described. As a result, there has been a quite considerable increase in the number of College members, and it is believed without any sacrifice of quality.

There were 903 members of the College listed in the 1941 Directory published in March of that year. Our records show that on October 1, 1941, the membership was 1,028, and on October 1, 1942, 1,197. On November 1, 1943, the membership was 1,479.

The membership as of today (November 10) is 1,485,* consisting of 1,172 Fellows, 133 Associate Fellows, and 180 Associate Members. There are 39 applications pending as of the same date. The records further show that 40 applications for membership in the College have been rejected.

It is axiomatic that if the College is to continue its activities, new blood must continuously be brought into the organization, and this is the function of the Membership Committee.

The procedure followed out by the Membership Committee received general approval of those consulted, and especially that of the President of the College. We have attempted to maintain an attitude of dignified approach to the individual rather than one of soliciting. In this connection, it seems to me that once having invited an individual to apply for membership in the College we should not, perhaps, give a bad impression by again extending an invitation unless some particular reason for the same should have arisen in the meantime. To press the issue by repeated invitations would seem to detract a bit from the dignity of the College. It is preferable to gradually afford the opportunity of membership to those who seem desirous of becoming members, rather than to put on a "campaign" for the purpose.

The question of including other professional groups through amalgamation between the College and such groups has arisen. This, although it might come under the head of new membership, is rather a matter of policy, and, therefore, to be decided by the Board of Regents. In any case, such procedure offers many difficulties and disadvantages and, in my opinion, the disadvantages would very considerably outweigh any gains made.

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*Membership, December 31, 1943—1,518.

The Membership Committee has at times received letters from individuals who have been invited to make application for membership to the effect that the American College of Chest Physicians is but a duplication of some other group. I doubt if this is actually the case. I personally know of no other society which has the same purpose, scope of activities, and aim as has the American College of Chest Physicians. As a matter of fact, some of the members of other societies would not meet our minimum requirements for admission to the College.

We must make the College so prominent and outstanding that no chest specialist can afford not to belong. Eventually, through the efforts of the College, all chest specialists who are members will be better informed, better qualified, and better recognized in their specialty as the result of the stimulation afforded by the association made possible through their membership.

It is desired to thank the Regents, Governors, and other members who have so wholeheartedly supported the Membership Committee and who have submitted lists of potential members. In conclusion, I wish to express the debt of gratitude the Committee owes the Executive Secretary, Mr. Murray Kornfeld, whose initiative, untiring energy and vision has contributed more than any other factor to whatever success the Membership Committee may have had.

Major General Shelley U. Marietta, F.C.C.P.
Chairman.

REPORT OF THE COUNCIL ON MILITARY AFFAIRS AND PUBLIC HEALTH*

In the May-June, 1943, issue of our journal, *Diseases of the Chest*, (Volume IX, Number 3) the Council on Military Affairs and Public Health of the American College of Chest Physicians published an article entitled "Tuberculosis in Our Industrial Army: An Appeal to Management and Labor." We assembled in the article certain facts and statistics with the hope that they would constitute an appeal to both labor and management for mass x-ray of industrial workers. The slogan adopted by our Council was: "Tuberculosis Is Preventable and Curable—So Put Every X-ray Machine to Work to Find Tuberculosis."

The Council authorized the printing of 5,000 reprints of this article which we thought would adequately serve our purpose. A letter was addressed to the members of the tuberculosis committees of the state and county medical societies throughout the country, advising them that copies of the reprints were available for distribution to industrial physicians and management. In less than two weeks, requests were received for more than the 5,000 copies of these reprints and a second printing of 5,000 copies was authorized. Letters were also mailed to a selected list of medical superintendents of tuberculosis sanatoria. Orders have been received in excess of 10,000 reprints authorized by the Council on Military Affairs and Public Health.** The Council was obliged to pro rate many of the requests for these reprints.

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*Presented before the Meeting of the Board of Governors and Regents, Cincinnati, Ohio, November 17, 1943.

**An additional printing of 10,000 copies has been authorized.

Requests for the reprints were received from physicians in thirty-three states and the District of Columbia. The largest amount, 1,046, were sent to New York State, and the least, 6, were sent to Arizona.

In view of the difficulty in securing new x-ray equipment, the Council has recommended that every available x-ray machine be utilized to its maximum capacity. The question of obtaining sufficient x-ray films was taken into consideration by the Council. It was felt that a greater economy could be effected by discovering and isolating active cases of tuberculosis.

The Council urges close cooperation between industry and the medical profession in helping to eliminate tuberculosis from our industrial army.

Dr. Charles M. Hendricks, F.C.C.P.
Chairman

REPORT OF THE NATIONAL COUNCIL OF TUBERCULOSIS COMMITTEES*

I am pleased to report that the National Council of Tuberculosis Committees has made good progress in having tuberculosis committees appointed in the state medical societies in this country. As of the present date, the District of Columbia and 41 states have established committees on tuberculosis in their state medical societies. The 7 states which have not as yet established tuberculosis committees in their state medical societies are: Alabama, Massachusetts, Mississippi, Ohio, Vermont, West Virginia and Wyoming. Letters have been received from the Governors of the College in Vermont and Wyoming, informing this Council that the necessary steps are being taken to establish committees on tuberculosis in their state medical societies. This would leave but 5 states in this country without a state tuberculosis committee. The states which have set up tuberculosis committees for the first time this year are: California, Kentucky, and Maryland.

Your Council is pleased to report that in California and in Kentucky, all of the members appointed to the tuberculosis committees are members of the American College of Chest Physicians. In Maryland, the Chairman of the committee and one other member are Fellows of the College.

The next important step in the program of this Council is to obtain the organization of similar committees on tuberculosis or diseases of the chest in as many of the county medical societies throughout the country as is possible. All of the county committees should, in their respective states, function under the direction of the state tuberculosis committees. This method of organization would enable a larger group of physicians to participate in the program for the control of tuberculosis. Other problems concerning diseases of the chest could be discussed at the state meetings and in the county societies. In those states where tuberculosis committees have been organized in the county medical societies, the members of these committees have been invited to attend the College Chapter meetings and urged to participate in the discussions.

There are 3,043 counties in the United States. While not all counties would be able to organize such committees, yet if a considerable number of

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*Presented before the Meeting of the Board of Governors and Regents, Cincinnati, Ohio, November 17, 1943.

these counties were to have tuberculosis committees as a part of their county medical societies, it would open up great possibilities for the presentation of programs concerning tuberculosis and other diseases of the chest. This is an added reason why the establishing of these committees should be stimulated even though many of the physicians are engaged in war activities.

Recently the Council on Military Affairs and Public Health of the College sent a reprint of an article entitled: "Tuberculosis in Our Industrial Army; An Appeal to Management and Labor" to members of the state and county tuberculosis committees of record at the Executive Offices of the College. As Dr. Hendricks, the Chairman of that Council, has reported to you this evening, requests were received at the Executive Offices of the College for more than 10,000 copies of this reprint. The members of these committees were requested to distribute the reprints to the plant physicians and accompany same with a letter urging the industrial establishments in their communities to x-ray all industrial workers. Several requests were received for literature for distribution to industrial workers. These requests were turned over to the state tuberculosis associations.

When the College undertook the establishing of tuberculosis committees in the state medical societies, there were fewer than 12 states which had set up tuberculosis committees as a part of their organization. This report will show how far we have progressed towards our ultimate objective. Your Council is proud to have had a part in the establishing of these tuberculosis committees.

Dr. James H. Stygall, F.C.C.P.
Chairman.

REPORT OF THE COUNCIL ON POSTGRADUATE MEDICAL EDUCATION*

A letter was sent to each member of the Council on Postgraduate Medical Education of the College offering the following recommendations:

1) That each member of the Council be assigned to a group of states and be asked to contact the chairmen and secretaries of the medical and surgical sections of their state and district medical societies and urge that papers dealing with diseases of the chest be placed on the programs at the annual meetings.

2) That members of the Council might suggest or recommend competent speakers for the presentation of such papers.

3) That the members of the Council contact the presidents and secretaries of the state and district Chapters of the College in the states assigned to them and offer their assistance in securing speakers on subjects for presentation at the meetings of these state and district chapters.

4) That the Council on Postgraduate Medical Education should, as a unit, contact the chairmen and secretaries of the scientific sections of the American Medical Association with the idea of securing places on the program for subjects concerning diseases of the chest.

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I wish to report that the first three of these recommendations were carried out by a number of members of the Council, but that no action was taken on recommendation number 4, due to the fact that the meeting of the American Medical Association for the year 1943 was cancelled.

Correspondence between members of the Council and officials of state and district medical societies was sent to the Executive Offices of the College to be kept as a permanent record of the activities of the Council.

On April 8, 1943, letters were directed to the Governors and Regents of the College recommending that a Speaker's Bureau be established. We asked for the names of members of the College who would be available as speakers for medical meetings. Recommendations were received from many of the Governors and Regents, and these names were prepared in mimeographed form at the Executive Offices of the College. Copies of the lists were furnished, upon request, to those societies that were interested in obtaining speakers.

The Council recommends that this Speaker's Bureau be enlarged and made a permanent part of the Council's activities and that the officers of the College Chapters and the officials of state and medical county societies be mailed copies of the names on the Speaker's Bureau. Most of the College chapters have held meetings during 1943 and scientific programs were presented at many of these meetings. Despite the difficulty of travel and the exigency of war, the Council on Postgraduate Medical Education recommends that state and district meetings be encouraged and if possible, a national meeting of the College be held during 1944 and that programs concerned with the latest advances in the specialty of diseases of the chest be presented at these meetings.

Dr. Edward P. Eglee, F.C.C.P.
Chairman

REPORT OF THE STATISTICAL COMMITTEE*

In July, 1942, I received notification from the President of the College that I was appointed as the Chairman of the Statistical Committee of the College and that the other members appointed to serve on this Committee were Dr. Forrest P. Baker, Oklahoma; Dr. Grover C. Bellinger, Oregon; Dr. Martin Collier, New Jersey, and Dr. Edwin R. Levine, formerly of New York City, and now with the Michael Reese Hospital of Chicago.

After communicating with the Executive Secretary of the College as directed by the President, questionnaires completed in 1940 from 263 hospitals and sanatoria were turned over to our Committee for study and tabulation.

These completed questionnaires were received from 110 county institutions, 35 state sanatoria, 17 municipal and city hospitals, 20 endowed institutions, 19 private sanatoria, 16 sanatoria maintained by church and religious organizations, 6 institutions maintained by fraternal associations, 14 veterans' hospitals, 9 army and navy hospitals, 8 Indian sanatoria, 8 preventoria, and 1 from an institution for the rehabilitation of the tuberculous.

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The 263 questionnaires were divided by districts and distributed to the members of the Committee for study. A letter of explanation as to how the information was to be compiled and tabulated accompanied the questionnaires. Our purpose in doing this was to arrange for a uniform method of compiling and recording this information so that when the reports were received from the members of the Committee, the total statistics could then be tabulated into a final report.

I am pleased to report that four members of the Committee have completed the work assigned to them and that we have received a report of progress from the fifth member of the Committee.

The questionnaires had four main subdivisions with a total of eighty questions. The first main division dealt with general information regarding the sanatoria and hospitals. The second main division concerned statistics for a five year study of various types of collapse therapy. The third main division related to salaries, maintenance, and administration for medical personnel. The fourth and final main division was concerned with questions relating to occupational therapy, vocational training and rehabilitation.

The Statistical Committee of the College is of the opinion that a great deal of useful information will be obtained from this survey. As early as possible, all of the material will be summed up in a final report which will be presented to the Board of Regents of the College and when approved, this report will be prepared and submitted for publication to the editor of our journal, *Diseases of the Chest*.

Dr. Otto C. Schlack, F.C.C.P.
Chairman.

OBITUARIES

LIEUT. COL. HUGH B. DISHAROON, M.C., A.U.S.

1910-1943

Hugh B. Disharoon, Lieutenant Colonel, Medical Corps, Army of the United States, died on August 22, 1943, at the Station Hospital, Fort Benning, Georgia.

Lieutenant Colonel Disharoon was born in Roanoke, Alabama, on September 4, 1910. After receiving the degree of A.B. (1932) from the Vanderbilt University, he entered the Medical School of that University and graduated in the class of 1935. After internships at the Vanderbilt Hospital and Cincinnati General Hospital, he returned to Vanderbilt for postgraduate work in obstetrics and gynecology. Upon completion of this postgraduate work he began a general practice in Lewisburg, Tenn.

On June 12, 1940, he accepted appointment as First Lieutenant, Medical Corps Reserve, and entered on active duty July 1, 1940. On July 12, 1941, he accepted an appointment in the Medical Corps of the Regular Army. He served at Fitzsimmons General Hospital, Denver, Colorado; Fort Knox, Kentucky; Camp Polk, Louisiana; Camp Young, California, and Fort Benning, Georgia.

He was married to Miss Virginia Evans, who survives him, as do two children, Martha Helen and Virginia Evans. His family resides at 1703 Walnut Street, Hopkinsville, Kentucky.

Major General S. U. Marietta, F.C.C.P.
Governor, U. S. Army Medical Corps

WILLIAM EMMETT DENMAN

1881-1943

Dr. William Emmett Denman was born in Tallhatchie County, Mississippi, November 4, 1881, the son of Dr. and Mrs. T. J. Denman. Dr. Denman obtained his early education in the schools of Tallhatchie County, later entering Mississippi College at Clinton, where he obtained his premedical training and received his liberal arts degree. He entered the University of Tennessee and graduated from this school in 1907. His medical career began at Philipp, Mississippi, as a general practitioner. He later moved to Roswell, New Mexico, and from there to Asheville, North Carolina, where he was associated with various groups in the treatment of diseases of the chest. Dr. Denman later did additional work at the Trudeau Sanatorium, Saranac Lake, New York.

In 1926 he moved to Greenwood, Mississippi, where he entered the private practice of medicine. He remained in Greenwood until his death from coronary occlusion on August 28, 1943.

Dr. Denman was an active member of the staff of the Greenwood Leflore Hospital. He was a member of the Presbyterian Church, the Chamber of Commerce, and was a Mason. He was a member of the Deta County Medical Society, Mississippi State Medical Association, American Medical Association, and a Fellow of the American College of Chest Physicians.

Dr. Denman was married to Miss Grace Leighton of Utica, Mississippi. In addition to the widow, he is survived by two sons and one daughter, Dr. W. E. Denman, Jr., Sanatorium, Mississippi; Lt. Jean K. Denman, 10th Air Force, U. S. A.; and Miss Dorothy Denman, Greenwood; four brothers, Richard Denman, Greenwood; Dr. C. W. Denman, Hughes, Arkansas; Kenneth A. Denman, Memphis, Tennessee; Edwin L. Denman, Winter Haven, Florida; three sisters, Mrs. William Seale, Cranshaw, Mississippi; Mrs. E. M. Sayle, Memphis, Tennessee, and Mrs. Mike Hey, Charleston, Mississippi.

Dr. John S. Harter, F.C.C.P.
Governor for Mississippi.

JOHN JOSEPH RANDALL

1892-1943

John Joseph Randall died on April 29th, 1943, at Wynantskill, New York. He was superintendent of the Pawling Sanatorium for 12 years. He graduated from the Albany Medical School in 1917 and interned at the Ellis Hospital, Schenectady, New York. He was commissioned a captain and served with the United States Medical Corps as a tuberculosis specialist in World War I. Upon his return from service he was appointed superintendent of the Sunny Acres Sanatorium, Cleveland, Ohio, leaving there in 1931 to accept the position as superintendent of the Pawling Sanatorium.

In 1938 he took postgraduate work in bronchoscopy at Chevalier Jackson Clinic at Temple University, after which he initiated a bronchoscopic clinic at the Pawling Sanatorium.

He was a member of the Rensselaer County Medical Society; New York State Medical Society; American Medical Association; National Tuberculosis Association; State Association of Superintendents of Tuberculosis

Hospitals; American Hospital Association; American Public Health Association; and a Fellow of the American College of Chest Physicians.

He is survived by his wife, the former Miss Ann E. Plunkett; two sisters, Mrs. Grace Dunn, New York, and Miss Helen Randall, Far Rockaway, L. I.; and one brother, David E. Randall, Cleveland, Ohio.

Nelson W. Strohm, F.C.C.P.
Governor for New York.

BOOK REVIEWS

Tuberculosis as It Comes and Goes, Edward W. Hayes, B.S., M.D., F.A.C.P., F.C.C.P., Monrovia, California. 187 Pages, 42 Illustrations. First Edition, 1943. Price: $2.00. Potts Memorial Institute, New York (National Tuberculosis Association).

This is a monograph intended to serve as a textbook for tuberculosis patients and their friends. It gives in brief form a general idea of the cause and nature of tuberculosis as well as a correct idea of its treatment.

The history, bacteriology, and epidemiology of tuberculosis are outlined, with emphasis on such practical considerations as the importance of early diagnosis by means of the tuberculin test and x-ray. The N. T. A. system of classification is presented, with admirable illustrative examples consisting of diagrammatic sketches of x-ray films. The Ornstein-Ulmar system also is discussed.

Dr. Hayes considers the subject of the treatment of secondary tuberculosis under six generalities which bear repeating: First, tuberculosis is an infectious disease which is not cured by climate, by diet, by heliotherapy, or by drugs. Second, while the disease is localized in the lungs, it involves the whole system, and as a systemic disease, it is accompanied by a depleted vitality and a general lowered resistance. Third, pulmonary tuberculosis jeopardizes the life of the patient, not as immediately, but just as surely as does acute appendicitis. Fourth, tuberculosis of the lungs is the most curable of chronic diseases. Fifth, patients who have this disease, generally speaking, regain their health through attention to details, or lose their lives through a failure to pay attention to details. Sixth, basically, the treatment of pulmonary tuberculosis consists of the outlining of a mode of life with attention to details for each patient and the guiding of each patient through the cure by close medical supervision.

The paragraphs on rest, the basic principle in treatment, are the best in the book. In powerful terms the author lays down the law regarding the reasons for physical and emotional rest. His great experience is obvious throughout, e.g., in his disposal of the class of patients who recover in spite of violation of accepted principles of treatment; "Dead men tell no tales." The discussion of diet, climate, and heliotherapy reflects the accepted viewpoints on the subjects.

Mechanical therapy as an adjunct to rest is the second general method of treatment and may be non-surgical or surgical. Of the latter the author outlines the simpler forms of collapse therapy, e.g., artificial pneumothorax, phrenic nerve interruption, intrapleural pneumonolysis, oleothorax, and the major forms, e.g., thoracoplasty, apicolysis, and extrapleural pneumothorax. These procedures are illustrated by sketches of x-rays, which give the patient a clear idea of the mechanism of the collapse.

The chapters on use of drugs, complications, pulmonary hemorrhage, and pregnancy and tuberculosis are characterized by uniformly sound logic. In the discussions on curability and prognosis an optimistic outlook is maintained, as befits a book prepared for laymen.

Prof. Laurence de Rycke has contributed an excellent chapter on suggestions to visitors, from the point of view of an ex-patient. In some respects it is more direct than a physician's reserve permits him to be, and it may be a valuable time saver in one's patient-visitor education program.

There is a bibliography and an index.

This book is highly recommended. It is remarkably free from statements likely to disturb the patient or lead him to question some detail in his regimen. The style, printing, and illustrations are attractive.

Sheldon Domm, M.D.

Synopsis of Allergy, Harry L. Alexander, A.B., M. D. 246 Pages, 22 Illustrations, 29 Tables. First Edition, 1941. C. V. Mosby, St. Louis.

In this admirable treatise on a difficult subject the editor of the Journal of Allergy presents the subject of allergy in terms of present-day thought. The plan of the book is that of a remarkably concise summary of the clinical expressions of human hypersensitiveness in all its forms. The generally accepted viewpoints are given, and where there is disagreement the author simply states his own interpretation.

In the brief historical sketch Dr. Alexander mentions the two aspects of allergy, the clinical and the immunologic. Allergic disorders are classified as non-bacterial including asthma and hay fever (atopy), contact dermatitis, physical allergy, drug allergy, and serum allergy, and bacterial, e.g., the tuberculin reaction.

Atopy is a disorder in which the individual inherits a predisposition to become hypersensitive to foreign non-bacterial substances (atopens). Symptoms of atopy result only from contact between such substances and tissues specifically sensitived to them. The three organs to which most atopic disturbances are related are the respiratory tract, the skin, and the gastrointestinal tract (the shock organs). The lesions of atopy, all of which are reversible, consist of edema, smooth muscle spasm, and hypersecretion from mucous glands. The mechanism of the production of atopic lesions appears to be release of histamine through stimulation of parasympathetic nerve endings. The most important diagnostic procedure in atopy is a history of contact with atopens. The three principles of treatment referable to all atopic disorders are protection against the offending atopen, hyposensitization, and drug therapy, the first being by far the most important. Epinephrine is the specific drug but ephedrine and synthetics such as neo-synephrine are of value.

The chapter on bronchial asthma should be of interest to every chest physician. In asthma the site of changes resides particularly in the bronchioles. During a paroxysm there is engorgement of the capillaries of the subepithelial layer followed by edema, spasm of the smooth muscle, and increased mucus secretion. In chronic asthma there is hypertrophy of the muscle layer and marked eosinophilic infiltration. Likewise there is hypertrophy of the mucous glands, producing excessive mucus which is the principal cause of obstruction to the smaller airways. The chief pathological change in the lung during a paroxysm is acute emphysema.

The chief change in chronic asthma is chronic emphysema. Death from bronchial asthma is rare. It occurs under two circumstances. One is accidental, coming immediately or soon after an overdose of an atopen in a patient extremely hypersensitive to it. The other is status asthmaticus which remains intractible to all treatment.

The most successful treatment of bronchial asthma is prophylactic. This includes detection of and prevention of contact with the offending atopen. The finer points in the use of epinephrine and aminophyllin, the drugs of choice for symptomatic relief, are of practical value to the reader. The importance of early treatment is stressed, in order to avoid the dread complication of emphysema. The discussion of the latter condition is of particular interest to chest physicians as it describes the early diagnosis as well as practical forms of treatment. The complications of chronic bronchitis and bronchiectasis are outlined.

The management of hay fever is lucidly presented, including the recognized forms of desensitization: preseasonal, perennial, and coseasonal. There are short chapters on atopic rhinitis, gastro-intestinal allergy, and headache, conjunctivitis, and other disorders.

The allergic dermatoses are of interest in view of the fact that skin lesions are by far the most common expressions of allergy, and reflect all forms of the disorder. Acute urticaria is usually due to atopy or to drug allergy. Chronic urticaria is usually of obscure etiology, and extrinsic allergins are rarely at fault. Treatment in both types is symptomatic. Allergic purpura (Henoch-Schonlein) is of some importance in differential diagnosis; it is usually due to hypersensitivity to foods. Atopic dermatitis is a disease of early life, skin tests are positive, and elimination measures are effective. Contact dermatitis is one of the most frequent diseases of man. It is purely an acquired type of hypersensitiveness, and the treatment is removal of the cause.

Physical allergy and drug allergy are briefly handled. The two types of serum allergy, serum accident and serum sickness, are described. Only two pages are devoted to the whole subject of bacterial allergy, but Dr. Alexander predicts that it promises to surpass in scope and importance all other forms of allergy. There is an index and a bibliography. The appendix contains directions for preparation of atopens, elimination diets, etc.

This book is a valuable addition to the library of any physician who wishes to keep abreast of current thought in a fast-moving branch of medicine closely related to his own specialty.

Sheldon Domm, M.D.